L13 1 S 2696-92-6 ← Cl. 4 NOCl

FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 14:40:10 ON 29 JUN 2002

FILE 'REGISTRY' ENTERED AT 14:40:26 ON 29 JUN 2002
SET SMARTSELECT ON
L14 SEL L13 1- CHEM : 10 TERMS
SET SMARTSELECT OFF

FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 14:40:27 ON 29 JUN 2002
L15 2409 S L14/BI
L16 0 S L15 AND HYPOXEMI?
L17 7 S L15 AND (LUNG# OR PULMONARY)

=>

=> d 1-7 bib ab

L17 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2002 ACS
AN 2001:582316 CAPLUS
DN 135:147442
TI Treating **pulmonary** disorders with gaseous agent causing repletion of GSNO
IN Stamler, Jonathan S.
PA Duke University, USA
SO U.S. Pat. Appl. Publ., 7 pp., Cont.-in-part of U.S. Ser. No. 390,215.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2001012834	A1	20010809	US 2001-782077	20010214
	US 6314956	B1	20011113	US 1999-390215	19990908
PRAI	US 1999-390215	A2	19990908		

AB **Pulmonary** disorders in which the GSNO pool or glutathione pool in the **lung** is depleted and where reactive oxygen species in **lung** are increased, are treated by delivering into the **lung** as a gas, agent causing repletion or increase of the GSNO pool or protection against toxicity and does so independently of reaction with oxygen. Agents include Et nitrite, NOCl, NOBr, NOF, NOCN, N2O3, HNO, and H2S. Optionally, N-acetylcysteine, ascorbate, H2S or HNO is administered in addn. to other GSNO repleting agent to potentiate the effect of said agent.

L17 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2002 ACS
AN 1987:570108 CAPLUS
DN 107:170108
TI Synergistic interaction between nitrogen dioxide and respirable aerosols of sulfuric acid or sodium chloride on rat **lungs**
AU Last, Jerold A.; Warren, Darren L.
CS California Primate Res. Cent., Univ. California, Davis, CA, 95616, USA
SO Toxicol. Appl. Pharmacol. (1987), 90(1), 34-42
CODEN: TXAPA9; ISSN: 0041-008X
DT Journal
LA English
AB Rats were exposed for 1, 3, or 7 days to 5 ppm NO2, alone or in combination with 1 mg/m3 H2SO4 or NaCl aerosols. The apparent rate of collagen synthesis by **lung** minces was measured after 7 days of exposure, and the protein content of whole **lung** lavage fluid was measured after 1 or 3 days of exposure. Responses from rats exposed to 5 ppm NO2 alone were significantly different from controls by these assays. A synergistic interaction was demonstrated between 5 ppm NO2 and 1 mg/m3 of either H2SO4 or NaCl aerosol as evaluated by measurement of the rate of **lung** collagen synthesis. A synergistic interaction was also demonstrated by the criterion of increased protein content of **lung** lavage fluid in rats exposed to 5 ppm NO2 and 1 mg/m3 H2SO4 aerosol after 1 day of exposure and between 5 ppm of NO2 and 1 mg/m3 NaCl aerosol after 3 days of exposure. These observations with 5 ppm NO2 alone and in combination with 1 mg/m3 NaCl aerosol support the hypothesis that formation of **nitrosyl chloride** may contribute to a synergistic interaction between NO2 gas and NaCl aerosol. Apparently, in general, combinations of oxidant gases with respirable acidic aerosols or with acidogenic gases demonstrate interactive effects on rat **lungs** . Such a hypothesis is testable and makes specific predictions about

effects of inhalation of pollutant mixts.

L17 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2002 ACS
AN 1959:85262 CAPLUS
DN 53:85262
OREF 53:15379c-h
TI The nature of the toxicity of some N-nitroso-N-(2-chloroethyl)carbamates in animals and man
AU Kramer, Stanley P.; Seligman, Arnold M.; Gaby, Samuel D.; Solomon, Robert D.; Miller, Jacob I.; Williamson, Charles; Witten, Benjamin
CS Sinai Hosp., Baltimore, MD
SO Cancer (1959), 12, 446-62
DT Journal
LA Unavailable
AB The following N-alkyl carbamates were prepd. by treatment of 2-naphthyl or phenyl chlorocarbonate with various amines: butyl N-(2-chloroethyl)carbamate, b. 73.degree. (11 mm.); phenyl N-(2-chloroethyl)carbamate, m. 71-2.degree.; 2-naphthyl N-(2-chloroethyl)carbamate, m. 137-8.degree.; 2-naphthyl N-ethylcarbamate, m. 127.5-8.5.degree.; 2-naphthyl N-(2-hydroxyethyl)carbamate, m. 138-40.degree.. The N-nitroso derivs. of the carbamates were prepd. by treating the compds. in glacial AcOH-Ac2O with approx. 140% excess **nitrosyl chloride**. The compds. thus made were: N-nitroso-N-(2-chloroethyl)propylamine, n24D 1.4704, b. 56-8.degree. (0.6-0.7 mm.); N-nitroso-N-(2-chloroethyl)propionamide, n24D 1.4707, b. 46-7 (0.82-0.85 mm.); ethyl N-nitroso-N-ethylcarbamate, n26D 1.4334, b. 78-80.degree. (24 mm.); butyl N-nitroso-N-(2-chloroethyl)carbamate (I), b. 60.degree. (11 mm.); phenyl N-nitroso-N-(2-chloroethyl)carbamate; 2-naphthyl N-nitroso-N-(2-chloroethyl)carbamate (II), m. 71-2.5.degree.. N-(2-Chloroethyl)propionamide, n25D 1.4635, b. 82-4.degree. (0.36-0.39 mm.), was also prepd. The susceptibility of the compds. to enzymic hydrolysis by normal and neoplastic tissue was detd. I was hydrolyzed to the greatest extent by liver from mice or dogs. Other more active tissues included mouse small intestine and dog kidney, although most tissues had some activity. For II the liver, pancreas, and kidney of both mice and dogs and dog **lung** showed the greatest activity. Both I and II were also hydrolyzed by various normal human tissues. In some cases human tumor tissue had greater hydrolytic ability than the corresponding normal tissue; in others there was less activity. Serums from humans, mice, dogs, and guinea pigs showed considerable hydrolytic activity toward I or II. Studies in mice and dogs showed that the compds. capable of being hydrolyzed to 2-chloro-1-diazoethane were the most toxic. In animals the N-nitroso-N-(2-chloroethyl)carbamates usually caused **pulmonary** edema, liver damage, and shrinkage of the spleen. In several cancer patients treated with I, intractable **pulmonary** edema and liver necrosis resulted.

L17 ANSWER 4 OF 7 MEDLINE
AN 91190289 MEDLINE
DN 91190289 PubMed ID: 2012683
TI Synergistic effects of air pollutants: ozone plus a respirable aerosol.
AU Last J A
CS Department of Internal Medicine, University of California, Davis.
SO RESEARCH REPORT / HEALTH EFFECTS INSTITUTE, (1991 Jan) (38) 1-32; discussion 33-43.
Journal code: 8812230. ISSN: 1041-5505.
Report No.: NASA-91190289.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals; Space Life Sciences
EM 199105
ED Entered STN: 19910602
Last Updated on STN: 19910602
Entered Medline: 19910516
AB Rats were concurrently exposed to mixtures of ozone or nitrogen dioxide and respirable-sized aerosols of sulfuric acid, ammonium sulfate, or sodium chloride, or to each pollutant individually. Their responses to such exposures were evaluated by various quantitative biochemical analyses of **lung** tissue or lavage fluids, or by morphometric analyses. Such studies were performed in the acute time frame, generally involving exposures of from one to nine days, depending on the assays used. Correlations between the biochemical and morphometric results were examined over a wide range of pollutant concentrations in the exposure chambers. Good correlations were found between the most sensitive biochemical indicators of **lung** damage--the protein content of **lung** lavage fluid or whole **lung** tissue and the rate of **lung** collagen synthesis--and the morphometric estimation of volume density or volume percent of the centriacinar **lung** lesion characteristically observed in animals exposed to ozone. Synergistic interaction between ozone and sulfuric acid aerosol was demonstrated to occur at environmentally relevant concentrations of both pollutants by several of the analytical methods used. Such interactions were demonstrated at concentrations of ozone as low as 0.12 parts per million (ppm)2 and of sulfuric acid aerosol at concentrations as low as 5 to 20 micrograms/m3. The acidity of the aerosol is a necessary (and apparently a sufficient) condition for such a synergistic interaction between an oxidant gas and a respirable aerosol to occur. A hitherto unexpected synergistic interaction between nitrogen dioxide and sodium chloride aerosol was found during these studies; it is hypothesized that this was due to formation of their acidic (anhydride) reaction product, **nitrosyl chloride**, in the chambers during exposure to the mixture. Preliminary experiments treating exposed animals in vivo with various free-radical scavengers suggested that dimethylthiourea, a hydroxyl-radical scavenger, might be protective against effects of ozone on rat **lungs**. This observation might have mechanistic implications, but further studies will be necessary to determine the significance of these findings.

L17 ANSWER 5 OF 7 MEDLINE
AN 87320370 MEDLINE
DN 87320370 PubMed ID: 3629590
TI Synergistic interaction between nitrogen dioxide and respirable aerosols of sulfuric acid or sodium chloride on rat **lungs**.
AU Last J A; Warren D L
NC ES-00628 (NIEHS)
HL-07013 (NHLBI)
RR-00169 (NCRR)
SO TOXICOLOGY AND APPLIED PHARMACOLOGY, (1987 Aug) 90 (1) 34-42.
Journal code: 0416575. ISSN: 0041-008X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198710
ED Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19871001

AB We examined interactions in rats between NO2 gas and respirable aerosols of sulfuric acid (H2SO4) or sodium chloride (NaCl). Rats were exposed for 1, 3, or 7 days to 5 ppm of NO2 gas, alone or in combination with 1 mg/m3 of H2SO4 or NaCl aerosols. The apparent rate of collagen synthesis by **lung** minces was measured after 7 days of exposure, and the protein content of whole **lung** lavage fluid was measured after 1 or 3 days of exposure. Responses from rats exposed to 5 ppm of NO2 alone were significantly different from controls by these assays. A synergistic interaction was demonstrated between 5 ppm of NO2 and 1 mg/m3 of either H2SO4 or NaCl aerosol as evaluated by measurement of the rate of **lung** collagen synthesis. A synergistic interaction was also demonstrated by the criterion of increased protein content of **lung** lavage fluid in rats exposed to 5 ppm of NO2 and 1 mg/m3 of H2SO4 aerosol after 1 day of exposure and between 5 ppm of NO2 and 1 mg/m3 of NaCl aerosol after 3 days of exposure. These observations with 5 ppm of NO2 alone and in combination with 1 mg/m3 of NaCl aerosol support the hypothesis that formation of **nitrosyl chloride** may contribute to a synergistic interaction between NO2 gas and NaCl aerosol. These results suggest that, in general, combinations of oxidant gases with respirable acidic aerosols or with acidogenic gases will demonstrate interactive effects on rat **lungs**. Such a hypothesis is testable and makes specific predictions about effects of inhalation of pollutant mixtures.

L17 ANSWER 6 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92160552 EMBASE
DN 1992160552
TI Global atmospheric change: Potential health effects of acid aerosol and oxidant gas mixtures.
AU Last J.A.
CS Pulmonary Division, Department of Internal Medicine, California Univ. School of Medicine,Davis, CA 95616, United States
SO Environmental Health Perspectives, (1991) 96/- (151-157).
ISSN: 0091-6765 CODEN: EVHPAZ
CY United States
DT Journal; Article
FS 046 Environmental Health and Pollution Control
LA English
SL English
AB Inhalation toxicology experiments in whole animals have demonstrated a remarkable lack of toxicity of sulfuric acid in the form of respirable aerosols, especially in rats and nonhuman primates. Thus, much of the current experimental emphasis has shifted to the evaluation of the potential health effects of acid aerosols as components of mixtures. Rats have been concurrently exposed to mixtures of ozone or nitrogen dioxide with respirable-sized aerosols of sulfuric acid, ammonium sulfate, or sodium chloride, or to each pollutant individually. Their responses to such exposures have been evaluated by various quantitative biochemical analysis of **lung** tissue or wash fluids ('lavage fluid') or by quantitative morphological methods ('morphometry'). Such studies have mainly been performed in the acute time frame due to the inherent limitations of the most sensitive assays available and have generally involved exposures for 1 to 9 days, depending on the assays used. Good correlations were found between the most sensitive biochemical indicators of **lung** damage (protein content of **lung** lavage fluid or whole **lung** tissue and **lung** collagen synthesis rate) and the exposure concentration of oxidant gas present alone or in mixtures with acidic aerosols showing interactive effects. Synergistic interaction between ozone and sulfuric acid aerosol was demonstrated to occur at

environmentally relevant concentrations of both pollutants by several of the analytical methods used in this study. Such interactions were demonstrated at concentrations of ozone as low as 0.12 ppm and of sulfuric acid aerosol at concentrations as low as 5 to 20 .mu.g/m3. The acidity of the aerosol is a necessary (and apparently a sufficient) condition for such a synergistic interaction between an oxidant gas and a respirable aerosol to occur. A hitherto unexpected synergistic interaction between nitrogen dioxide and sodium chloride aerosol was found during these studies; it is hypothesized that this was due to formation of their acidic (anhydride) reaction product, **nitrosyl chloride**, in the chambers during exposure to the mixture.

L17 ANSWER 7 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 87211417 EMBASE
DN 1987211417
TI Synergistic interaction between nitrogen dioxide and respirable aerosols of sulfuric acid or sodium chloride on rat **lungs**.
AU Last J.A.; Warren D.L.
CS Department of Internal Medicine, University of California, Davis, CA 95616, United States
SO Toxicology and Applied Pharmacology, (1987) 90/1 (34-42).
ISSN: 0041-008X CODEN: TXAPA
CY United States
DT Journal
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
046 Environmental Health and Pollution Control
052 Toxicology
LA English
AB We examined interactions in rats between NO2 gas and respirable aerosols of sulfuric acid (H2SO4) or sodium chloride (NaCl). Rats were exposed for 1, 3, or 7 days to 5 ppm of NO2 gas, alone or in combination with 1 mg/m3 of H2SO4 or NaCl aerosols. The apparent rate of collagen synthesis by **lung** minces was measured after 7 days of exposure, and the protein content of whole **lung** lavage fluid was measured after 1 or 3 days of exposure. Responses from rats exposed to 5 ppm of NO2 alone were significantly different from controls by these assays. A synergistic interaction was demonstrated between 5 ppm of NO2 and 1 mg/m3 of either H2SO4 or NaCl aerosol as evaluated by measurement of the rate of **lung** collagen synthesis. A synergistic interaction was also demonstrated by the criterion of increased protein content of **lung** lavage fluid in rats exposed to 5 ppm of NO2 and 1 mg/m3 of H2SO4 aerosol after 1 day of exposure and between 5 ppm of NO2 and 1 mg/m3 of NaCl aerosol after 3 days of exposure. These observations with 5 ppm of NO2 alone and in combination with 1 mg/m3 of NaCl aerosol support the hypothesis that formation of **nitrosyl chloride** may contribute to a synergistic interaction between NO2 gas and NaCl aerosol. These results suggest that, in general, combinations of oxidant gases with respirable acidic aerosols or with acidogenic gases will demonstrate interactive effects on rat **lungs**. Such a hypothesis is testable and makes specific predictions about effects of inhalation of pollutant mixtures.

=>

NOCl N_2O_3

FILE 'CAPLUS' ENTERED AT 15:19:11 ON 29 JUN 2002
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FILE 'WPIDS' ENTERED AT 15:19:11 ON 29 JUN 2002
COPYRIGHT (C) 2002 THOMSON DERWENT

FILE 'MEDLINE' ENTERED AT 15:19:11 ON 29 JUN 2002

FILE 'EMBASE' ENTERED AT 15:19:11 ON 29 JUN 2002
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=> s (l15 or l23) and (asthma? or cystic fibro? or ard or adult respiratory distress or pneumon? or interstitial lung disease#)
L36 1 (L15 OR L23) AND (ASTHMA? OR CYSTIC FIBRO? OR ARD OR ADULT RESPIRATORY DISTRESS OR PNEUMON? OR INTERSTITIAL LUNG DISEASE#)

=> d

L36 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
AN 2001:582316 CAPLUS
DN 135:147442
TI Treating pulmonary disorders with gaseous agent causing repletion of GSNO
IN Stamler, Jonathan S.
PA Duke University, USA
SO U.S. Pat. Appl. Publ., 7 pp., Cont.-in-part of U.S. Ser. No. 390,215.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2001012834	A1	20010809	US 2001-782077	20010214
	US 6314956	B1	20011113	US 1999-390215	19990908
PRAI	US 1999-390215	A2	19990908		

=>

Cl. 5

N_2O_3

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=> d que l26
L21             1 SEA FILE=REGISTRY 10544-73-7
L22               SEL  L21 1- CHEM :      12 TERMS
L23          1279 SEA L22/BI
L25            13 SEA L23 AND (LUNG# OR PULMONARY)
L26            13 DUP REM L25 (0 DUPLICATES REMOVED)


=> d his l21-

     (FILE 'REGISTRY' ENTERED AT 14:49:21 ON 29 JUN 2002)
L21             1 S 10544-73-7

     FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 14:51:50 ON 29 JUN 2002

     FILE 'REGISTRY' ENTERED AT 14:52:32 ON 29 JUN 2002
                 SET SMARTSELECT ON
L22           SEL L21 1- CHEM :      12 TERMS
                 SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 14:52:33 ON 29 JUN 2002
L23          1279 S L22/BI
L24             0 S L23 AND HYPOXEM?
L25            13 S L23 AND (LUNG# OR PULMONARY)
L26            13 DUP REM L25 (0 DUPLICATES REMOVED)
```

L26 ANSWER 1 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 2001:582316 CAPLUS
DN 135:147442
TI Treating **pulmonary** disorders with gaseous agent causing repletion of GSNO
IN Stamler, Jonathan S.
PA Duke University, USA
SO U.S. Pat. Appl. Publ., 7 pp., Cont.-in-part of U.S. Ser. No. 390,215.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2001012834	A1	20010809	US 2001-782077	20010214
	US 6314956	B1	20011113	US 1999-390215	19990908
PRAI	US 1999-390215	A2	19990908		

AB **Pulmonary** disorders in which the GSNO pool or glutathione pool in the **lung** is depleted and where reactive oxygen species in **lung** are increased, are treated by delivering into the **lung** as a gas, agent causing repletion or increase of the GSNO pool or protection against toxicity and does so independently of reaction with oxygen. Agents include Et nitrite, NOCl, NOBr, NOF, NOCN, N2O3, HNO, and H2S. Optionally, N-acetylcysteine, ascorbate, H2S or HNO is administered in addn. to other GSNO repleting agent to potentiate the effect of said agent.

L26 ANSWER 2 OF 13 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-082275 [11] WPIDS
CR 2001-264980 [15]
DNC C2002-024794
TI Hemoglobin colloid composition, which increases cardiac output without affecting heart rate, includes, e.g., a hemoglobin-lipid coacervate and a dihydropyridine compound.
DC B05
IN ROONEY, M W
PA (ROON-I) ROONEY M W
CYC 1
PI US 2001034323 A1 20011025 (200211)* 29p
ADT US 2001034323 A1 CIP of US 1992-849610 19920311, Div ex US 1995-480189 19950607, US 2000-727170 20001130
FDT US 2001034323 A1 Div ex US 6187744
PRAI US 1995-480189 19950607; US 1992-849610 19920311; US 2000-727170 20001130
AB US2001034323 A UPAB: 20020215
NOVELTY - A combination of a hemoglobin (Hb)-based material and a guanosine 3',5'-cyclic monophosphate (cGMP) generating compound is used in a Hb colloid composition (HCC), and in a treatment method, for hemodilution, blood substitution, plasma expansion or fluid resuscitation.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for:

(A) HCC for use in hemodilution, blood substitution, plasma expansion or fluid resuscitation, comprising a Hb-based material and a guanosine 3',5'-cyclic monophosphate (cGMP) generating compound. The HCC is used for the purpose of producing oxygen delivery, in vivo, that is superior to that obtained with a Hb-based material alone. The HCC is free of adverse hemodynamic effects. The HCC selectively affects afterload relative to preload. The HCC intentionally increases stroke volume and cardiac output with no effect on heart rate;

(B) treatment of diseases or conditions which require a hemodiluent,

blood substitute, plasma expander or resuscitative fluid, comprising: (a) hemodiluting the patient by replacing red cell mass with a Hb-based material which is natural Hb, a Hb-lipid coacervate, a polymerized Hb molecule, a Hb conjugates or a covalent Hb tetramer; and (b1) administering, parenterally or enterally, a nitrovasodilator compound; (b2) administering, by inhalation, a nitrovasodilator which is nitric oxide, nitrous oxide, nitrogen dioxide, **nitrogen trioxide** or nitrogen tetroxide; and/or (b3) administering, enterally or parenterally, a cGMP generating compound which is a nitric oxide donor, a nitric oxide substrate, a nitric oxide synthase potentiator and/or a guanylate cyclase potentiator.

ACTIVITY - Hypertensive.

MECHANISM OF ACTION - Guanosine 3',5'-cyclic monophosphate generator; nitric oxide synthase potentiator; guanylate cyclase potentiator.

USE - The composition and process described above are useful for treatment of diseases or conditions which require a hemodiluent, a blood substitute, a plasma expander or a resuscitative fluid. Such conditions include, e.g., hypertension or anaphylactic shock.

ADVANTAGE - The HCC provides greater whole body oxygen delivery than that achieved by the Hb-based material alone. It also provides greater whole body oxygen delivery than that achieved by albumin, plasma protein fraction, serum,other plasma-derived colloids or synthetic colloids. It does not cause a negative effect on **pulmonary** or respiratory function, on cardiac function or on regional organ or tissue hemodynamic function.

Dwg.0/5

L26 ANSWER 3 OF 13 MEDLINE
AN 2002126707 IN-PROCESS
DN 21851714 PubMed ID: 11860912
TI **Lung** injury caused by passive smoking and its effects on cytokines in rats.
AU Pang B; Wang C; Weng X; et Al
CS Red Cross Chaoyang Hospital, Beijing 100020, China.
SO CHUNG-HUA YU FANG I HSUEH TSA CHIH [CHINESE JOURNAL OF PREVENTIVE MEDICINE], (2000 Mar) 34 (2) 104-5.
Journal code: 7904962. ISSN: 0253-9624.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA Chinese
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20020226
Last Updated on STN: 20020226
AB OBJECTIVE: A rat model with chronic bronchitis was replicated by passive inhalation of cigarette smoking fume to study its long-term effects on **lung** injury and nitric oxide (NO), interluekin-6 (IL-6), interleukin-8 (IL-8). METHODS: Levels of nitrogen dioxide (NO(2)) and **nitrogen trioxide** (NO(3)) were measured with spectrophotometry in rats indicating their level of nitric oxide (NO). Levels of IL-6 and IL-8 were determined by enzyme-linked immunosorbent assay (ELISA). RESULTS: Levels of NO in serum, bronchial alveolar lavage fluid (BALF) and **lung** tissue in the smoking group were significantly lower than those in the normal controls ($P < 0.01$). But, levels of IL-6 and IL-8 were higher in the smoking group than those in the controls. CONCLUSION: Long-term passive smoking could cause injury of **lung** tissue to certain extent, reduction in secretion of NO in endothelial cells and damage to **pulmonary** vessels.

L26 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2002 ACS

AN 1997:675540 CAPLUS
DN 127:322837
TI Procedure and device for minimizing risks in nitric oxide inhalation therapy
IN Eschwey, Manfred; Hege, Klaus; Krebs, Christian
PA Messer Griesheim G.m.b.H., Germany
SO Ger. Offen., 4 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 19612289	A1	19971002	DE 1996-19612289	19960328
	ZA 9702728	A	19971023	ZA 1997-2728	19970327
PRAI	DE 1996-19612289	A	19960328		

AB During therapy of **pulmonary** disorders by inhalation of a gas mixt. contg. NO, contaminating gases such as toxic NO2, N2O3, and N2O4 are removed by placing a filter comprising a S-contg. polymer in the gas stream. If the gas is humidified, the filter can also remove toxic NO2- and NO3- in the condensate and prevent their formation. A respirator incorporating such a filter is described with the aid of a schematic diagram.

L26 ANSWER 5 OF 13 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 97356153 EMBASE
DN 1997356153
TI Environmental health criteria for nitrogen oxides.
AU Dobson S.
CS Dr. S. Dobson, Institute of Terrestrial Ecology, Monks Wood Experimental Station, Abbots Ripton, Huntingdon, Cambridgeshire, United Kingdom
SO Environmental Health Criteria, (1997) -/188 (1-550).
ISSN: 0250-863X CODEN: EHCRDN
CY Switzerland
DT Journal; General Review
FS 046 Environmental Health and Pollution Control
LA English
SL English
AB Nitrogen oxides can be present at significant concentrations in ambient air and in indoor air. The types and concentrations of nitrogenous compounds present can vary greatly from location to location, with time of day, and with season. The main sources of nitrogen oxide emissions are combustion processes. Fossil fuel power stations, motor vehicles and domestic combustion appliances power stations, motor vehicles and domestic combustion appliances emit nitrogen oxides, mostly in the form of nitric oxide (NO) and some (usually less than about 10%) in the form of nitrogen dioxide (NO2). In the air, chemical reactions occur that oxidize NO to NO2 and other products. There are also biological processes that liberate nitrogen species from soils, including nitrous oxide (N2O). Emissions of N2O can cause perturbation of the stratospheric ozone layer. Human health may be affected when significant concentrations of NO2 or other nitrogenous species, such as peroxyacetyl nitrate (PAN), nitric acid (HNO3), nitrous acid (HNO2), and nitrated organic compounds, are present. In addition, nitrates and HNO3 may cause health effects and significant effects on ecosystems when deposited on the ground. The sum of NO and NO2 is generally referred to as NO(g). Once released into the air, NO is oxidized to NO2 by available oxidants (particularly ozone, O3). This happens rapidly under some conditions in outdoor air; in indoor air, it is generally a much slower process. Nitrogen oxides are controlling percursor

of photochemical oxidant air pollution resulting in ozone and smog formation; interactions of nitrogen oxides (except N2O) with reactive organic compounds and sunlight form ozone in the troposphere and smog in urban areas. NO and NO2 may also undergo reactions to form a range of other oxides of nitrogen, both indoor and outdoor air, including HNO2, and HNO3, **nitrogen trioxide** (NO3), dinitrogen pentoxide (N2O5), PAN and other organic nitrates. The complex range of gas-phase nitrogen oxides is referred to as NO(y). The partitioning of oxides of nitrogen among these compounds is strongly dependent on the concentrations of other oxidants and on the meteorological history of the air. HNO3 is formed from the reaction of OH- and NO2. It is a major sink for active nitrogen and also a contributor to acidic deposition. Potential physical and chemical sinks for HNO3 include wet and dry deposition, photolysis, reaction with OH radicals, and reaction with gaseous ammonia to form ammonium nitrate aerosol. PANs are formed from the combination of organic peroxy radicals with NO2. PAN is the most abundant organic nitrate in the troposphere and can serve as a temporary reservoir to reactive nitrogen, which may be regionally transported. The NO3 radical, a short-lived NO(y) species that is formed in the troposphere primarily by the reaction of NO2 with O3, undergoes rapid photolysis in daylight or reaction with NO. Appreciable concentrations are observed during the night. N2O5 is primarily a night-time constituent of ambient air as it is formed from the reaction of NO3 and NO2. In ambient air, N2O5 reacts heterogeneously with water to form HNO3, which in turn is deposited. N2O is ubiquitous because it is a product of natural biological processes in soil. It is known, however, to be involved in any reactions in the troposphere. N2O participates in upper atmospheric reactions contributing to stratospheric ozone (O3) depletion and is also a relatively potent greenhouse gas that contributes to global warming. 1.1.1 Atmospheric transport. The transport and dispersion of the various nitrogenous species in the lower troposphere is dependent on both meteorological and chemical parameters. Advection, diffusion and chemical transformations combine to dictate the atmospheric residence times. In turn, atmospheric residence times help determine the geographic extent of transport of given species. Surface emissions are dispersed vertically and horizontally through the atmosphere by turbulent mixing processes that are dependent to a large extent on the vertical temperature structure and wind speed. As the result of meteorological processes, NO(k) emitted in the early morning hours in an urban area typically disperses vertically and moves downwind as the day progresses. On sunny summer days, most of the NO(x) will have been converted to HNO3 and PAN by sunset, with concomitant formation of ozone. Much of the HNO3 is removed by deposition as the air mass is transported, but HNO3 and PAN carried in layers aloft (above the nighttime inversion layer but below a higher subsidence inversion) can potentially be transported long distances in oxidant-laden air masses. 1.1.2 Measurement. There are a number of methods available to measure airborne nitrogen-containing species. This document briefly covers methodologies currently available or in general use for in situ monitoring of airborne concentrations in both ambient and indoor environments. The species considered are NO, NO2, NO(x), total reactive odd nitrogen (NO(y)), PAN and other organic nitrates, HNO3, HNO2, N2O5, the nitrate radical, NO3/-, and N2O. Measuring concentrations of nitrogen oxides is not trivial. While a straightforward, widely available method exists for measuring NO (the chemiluminescent reaction with ozone), this is an exception for nitrogen oxides. Chemiluminescence is also the most common technique used for NO2; NO2 is first reduced to NO. Unfortunately, the catalyst typically used for the reduction is not specific, and has various conversion efficiencies for other oxidized nitrogen compounds. For this reason, great care must be taken in interpreting the results of the common chemiluminescence analyser in terms

of NO2, as the signal may include many other compounds. Additional difficulties arise from nitrogen oxides that may partition between the gaseous and participate phases both in the atmosphere and in the sampling procedure. 1.1.3 Exposure. Human and environmental exposure to nitrogen oxides varies greatly from indoors to outdoors, from cities to the countryside, and with time of day and season. The concentrations of NO and NO2 typically present outdoors in a range of urban situations are relatively well established. The concentrations encountered indoors depend on the specific details of the nature of combustion appliances, chimneys and ventilation. When unvented combustion appliances are used for cooking or heating, indoor concentrations of nitrogen oxides typically greatly exceed those existing outside. Recent research has shown in these circumstances that HNO2 can reach significant concentrations. One report showed that HNO2 can represent over 10% of the concentrations usually reported as NO2.

L26 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 1984:591192 CAPLUS
DN 101:191192
TI Studies on synthesis and anticancer activity of selected N-(2-fluoroethyl)-N-nitrosoureas
AU Johnston, Thomas P.; Kussner, Conrad L.; Carter, Ronald L.; Frye, Jerry L.; Lomax, Nancita R.; Plowman, Jacqueline; Narayanan, V. L.
CS South. Res. Inst., Kettering-Meyer Lab., Birmingham, AL, 35255-5305, USA
SO J. Med. Chem. (1984), 27(11), 1422-6
CODEN: JMCMAR; ISSN: 0022-2623
DT Journal
LA English
OS CASREACT 101:191192
AB Aminolysis of FCH2CH2N(NO)CO2C6H4NO2-2 with H2NCH2CH2OH, 1.alpha.,2.beta.,3.alpha.-2-amino-1,3-cyclohexanediol, cis-1,2-aminocyclohexanol, and 2-amino-2-deoxy-D-glucose gave the corresponding H2O-sol ureas, e.g., I. The H2O-insol. glutarimide analog II was prepd. by nitrosation of the corresponding urea. In trials with B16 melanoma and Lewis **lung** carcinoma the compds. were comparable to their Cl analogs as inhibitors; I seemed to be the most effective.

L26 ANSWER 7 OF 13 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 78381729 EMBASE
DN 1978381729
TI The higher oxides of nitrogen: inhalation toxicology.
AU Lamont Guidotti T.
CS Dept. Environm. Hlth Scis, Johns Hopkins Sch. Hyg. Publ. Hlth, Baltimore, Md. 21205, United States
SO Environmental Research, (1978) 15/3 (443-472).
CODEN: ENVRAL
CY United States
DT Journal
FS 037 Drug Literature Index
017 Public Health, Social Medicine and Epidemiology
046 Environmental Health and Pollution Control
LA English
AB The higher oxides of nitrogen (NO, NO2, and higher valence) are highly reactive compounds encountered in a variety of occupational exposures and are principal constituents of photochemical air pollution. Their chemical properties result in direct oxidation, free radical formation, nitrosation, nitrite ion release, and paramagnetic interactions with heme. NO is formed from the oxidation of atmospheric N2 in the internal

combustion engine and is converted to NO2, the compound of greater toxicity. Inhalation of NO2 in high concentrations may result in a triphasic sequence of acute bronchospasm, delayed **pulmonary** edema, and late bronchiolitis obliterans. Low concentrations appear to induce **pulmonary** fibrosis with chronic exposure and to inhibit **pulmonary** defense mechanisms, particularly macrophage function and ciliary motility. Animal and human population studies suggest that the greatest risk from low-dose-term exposure is reduced host resistance to viral and bacterial respiratory tract infections. The present national ambient air quality standard does not provide a large safety margin for this latter effect and should be reviewed.

L26 ANSWER 8 OF 13 WPIDS (C) 2002 THOMSON DERWENT
AN 1976-40374X [22] WPIDS
TI 1,3-Bis 2-chloroethyl-1-nitroso urea prepn - by reacting bis (2-chloroethyl) urea with excess **dinitrogen trioxide**.
DC B05
PA (USSH) US SEC DEPT HEALTH
CYC 8
PI DE 2528365 A 19760520 (197622)*
SE 7504719 A 19760608 (197626)
JP 51056414 A 19760518 (197627)
FR 2291187 A 19760716 (197638)
GB 1469381 A 19770406 (197714)
US 4028410 A 19770607 (197724)
CH 598204 A 19780428 (197819)
CA 1082223 A 19800722 (198032)
PRAI US 1974-523334 19741113
AB DE 2528365 A UPAB: 19930901
1,3-Bis(2-chloroethyl)-1-nitroso urea of formula (I) is prepd. by reacting 1,3-bis(2-chloroethyl)urea (II) with excess N2O3 at 0 to -20 degrees C in the presence of an organic solvent: (I) is a known medicament used as chemotherapeutic agent against cancer. Its activity is antineoplastic and it may be used against fast-growing cells in brain-tumours, cancer of the colon and **lung**. Hodgkins disease and multiple myeloma. The process is simpler and gives higher yields and purity than previously known ones.

L26 ANSWER 9 OF 13 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 74127746 EMBASE
DN 1974127746
TI [Goal directed prevention after exposure to nitrous gases].
GEZIELTE PRAVENTION NACH EXPOSITION DURCH NITROSE GASE.
AU Buhlmeyer G.
CS Bayer. Landesinst. Arbeitsmed., Nurnberg, Germany
SO Therapiewoche, (1973) 23/45 (4308-4312).
CODEN: THEWA6
DT Journal
FS 024 Anesthesiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
LA German
AB Nitrous gases in the respiratory air may be the cause of **pulmonary** edema producing severe, often fatal, clinical pictures after a latent period of 3 to 4 hr, rarely up to 24 hr. The potential dangers due to nitrous gases are from catalytic combustion of ammonia (nitric acid manufacture), the use of nitric acid in the chemical and explosives industry (nitration processes, manufacture of fertilizers, etc.), use of nitric acid in the metal industry (cleaning, pickling, etching, separation of precious metals), spilling of nitric acid, breaking of storing or

transport containers, burning of nitrocellulose, detonation and deflagration of nitroexplosives, oxidation of the atmospheric nitrogen at high temperatures (production of potassium nitrate according to the Norge method, welding, burning), and fermentation formation of nitrous oxides in silos (silo filler's disease). The higher oxidation stages of nitrogen, nitrogen dioxide (NO2), nitrogen tetroxide (N2O4), and **nitrogen trioxide** (**N2O3**) play a decisive role in the genesis of **pulmonary** edema. Treatment should be started as soon as possible. Therapeutic measures are indicated.

L26 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 1971:51419 CAPLUS
DN 74:51419
TI Influence of inert gases of the alveolar-arterial oxygen-difference
AU Liese, Wilfried; Muysers, K.; Pichotka, J. P.
CS Physiol. Inst., Univ. Bonn, Bonn, Ger.
SO Pfluegers Arch. (1970), 321(4), 316-31
CODEN: PFLABK
DT Journal
LA German
AB The alveolar and inspired O2 partial pressure was detd. in 10 healthy persons (age 20-35 years) during breathing of 20.9% O2 in different inert gases by continuous mass spectrometric anal.; the arterial O2 pressure was detd. by means of microelectrodes with arterial blood from the ear lobe. Mean alveolar arterial O2 pressure difference was 8.7 torr for N2O3, 15.3 torr for He-O2, and 16.3 torr for Ar-O2.

L26 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 1969:516271 CAPLUS
DN 71:116271
TI Health hazards in gas and electrowelding
AU Hoschek, Rudolf
SO Waerme (1969), 75(2-3), 82-5
CODEN: WARMAP
DT Journal
LA German
AB In gas welding, burning and explosion hazards are important. The most frequent accident causes are gas bottle explosions, the fall of gas bottles, etc. The C2H2 leaks are dangerous because of the presence of PH3 in industrial C2H2. A poorly adjusted flame can produce CO. The greatest danger in gas welding is the formation of NO, NO2, N2O3, and N2O4 by the combination of the N of the air with the O of the gas bottle at 3000.degree.. A good ventilation is needed because the presence of these N oxides can diminish the auto-purifying process of the **lungs** and can cause asphyxia. Pb and Cd aerosols formed by the heating of the protective layer are dangerous. During the electrowelding a smoke evolves that contains Fe2O3, amorphous SiO2, MnO, and even CaO and F in the case of basic calcareous electrodes. The amt. of Fe in the blood increases by inhalation of this smoke and electrowelders complain of nausea, fatigue, and giddiness. The consummation of alc., tobacco, accentuates these effects.

L26 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 1920:4857 CAPLUS
DN 14:4857
OREF 14:904g-i,905a
TI The existence of **nitrous anhydride** in the gaseous state
AU Wourtzel, Eugene

SO Compt. rend. (1920), 170, 109-11
DT Journal
LA Unavailable
AB The present state of knowledge concerning the existence of N2O3 as a gas is contradictory. Ramsay and Cundall (J. Chem. Soc. 57, 591 (1885)), and **Lunge** and Porschnew (Z. anorg. Chem. 7, 294(1894)) by vapor density measurements came to the conclusion that there was complete dissociation into NO and NO2. On the other hand LeBlanc (Z. Elektrochem. 12, 270(1906)) found that NO and NO2 in a gaseous mixt. were absorbed in equiv. amts. to form almost exclusively the nitrite, while NO alone does not react and NO2 alone gives rise to equiv. amts. of nitrite and nitrate. In order to bring other evidence to bear the author in this note describes a study of the contraction produced upon mixing known amts. of NO and O2, keeping the former gas in excess. Using the app. and method previously employed (C. A. 14, 667) the contraction Pc is detd. by the relation Pc = PNO + PO2 - PM2, where the last term is the final pressure of NO in the volumeter. If NO2 alone is formed the game contraction can be calcd from Pc = (I + X)PO2 and K = 4PO2(I - X)2/x. However, Pc and P'c differ by about 3.3 and this discrepancy must be due to the formation of some N2O3-about 2.5 parts per 100. The const. K' = P'NOPNO2/PN2O3 is about 1100. The small quantity accounts for the failure to recognize N2O3 in vapor density measurements; and the reaction upon absorption in alkali is accounted for by the continuous formation of N2O3 from NO and NO2 as absorption proceeds and the equil. is displaced.

L26 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 1908:1074 CAPLUS
DN 2:1074
OREF 2:303a-i,304a-i,305a-i,306a-i,307a-d
TI On Determination of the Oxides of Nitrogen and the Theory of the Chamber Process
AU Lunge, G.; Berl, E.
SO Z. angew. Chem. (1908), 20, 1713-22
DT Journal
LA Unavailable
AB The most important point of difference between Raschig and the authors relates to the analytical methods for determination of the nitrogen oxides. As to those relating to the composition of **nitrogen trioxide, N2O3**, whether one considers it as a distinct chemical compound or as a mixture of NO and NO2 or N2O4, Raschig agrees with them that in presence of oxygen cone. H2SO4, when used as absorbent, gives correct results, while NaOH gives false ones. For the determination of N2O4 in presence of oxygen, Raschig considers cone. H2SO4 useless as absorbent, because of losses due to formation of ozone and nitrogen oxides of inactive form, so that the ratio of N2O3 to N2O5 appears not equal to, but greater than 1. He persists in his opinion that NaOH is the proper absorbent, permitting perfect absorption, and demonstrating correctly the breaking down of N2O4 into equal parts of nitrate and nitrite. In their earlier paper (Ibid., 19, 809) they conclusively showed that Raschig's statements as to the action of H2SO4 upon N2O4 were incorrect. As to the total absorption, as well as to the splitting up into N2O3+N2O5 they found that cone. H2SO4 gives figures satisfactorily close to the theoretical. As to the asserted splitting off of N2O, N2 and O3, Raschig ignores their manometric experiments (Ibid., 319, 817) in sharp contradiction to his statements. To prove his assertion that ozone is formed, Raschig mixes nitric oxide and air, gives the gases time to oxidize, and thin passes them into cone. H2SO4, "isonitrogen pentoxide" is completely absorbed, and the gases leaving the absorption vessel are passed into KI solution, precipitating iodine, and giving the solution an alkaline reaction, which

he ascribes as due to the formation of ozone. In an earlier experiment (Ibid. 18, 1288) a mixture of N_2O_5 and O, or, as Raschig would say, "Isonitrogenheptoxide," which is claimed to possess the same peculiarities of absorption as the pentoxide, was passed first into cone. H_2SO_4 and then into KI solution. In this case, however, the "solution remained perfectly clear." Therefore, at that time, no ozone formation took place, in contrast to the result in the later experiment. The results, therefore, of one or other of the 1905 or 1907 experiments must be wrong. Again, contrary to Raschig's views, they proved that while NaOH quantitatively absorbed N_2O_4, in the presence of oxygen only, the ratio of $NaNO_3$ to $NaNO_2$ formed, is greater than 1:1 while in the presence of a neutral gas, viz., nitrogen, the ratio is indeed as 1 : 1. As Raschig considers their results were vitiated by the presence of water, facilitating the formation of HNO_3, the authors prepared most carefully, thoroughly dry N_2O_4, as follows: 1. Preparation and Analysis of Pure Nitrogen Peroxide. Pure, dry lead nitrate was heated in combustion tubing, and the gases produced were condensed in an absorbing vessel, surrounded by a freezing mixture. The product thus obtained was twice fractionated in an all-glass apparatus in a stream of oxygen dried by P_2O_5, the gaseous mixture passing over P_2O_5, and the N_2O_4, then being condensed by a freezing mixture. The product thus obtained was again fractionated, rejecting first and last portions in another all-glass apparatus, the peroxide being in this case carried over by the stream of P_2O_5 dried oxygen without application of direct heat, through and over P_2O_5, and condensed in glass bulbs by a freezing mixture, and the glass bulbs then sealed up for weighing. Experiments with this pure, dry N_2O_4. were then made in the same way as their earlier experiments, taking every conceivable precaution to avoid contamination with water. The results are given in table form. They found that the mean ratio of N_2O_5 :N_2O_5, using H_2SO_4, as absorbent, and with or without free oxygen present, was 49:51, or as close to the theoretical: 50:50 as one can expect, cording to Raschig, owing to the splitting off of the oxygen, this ratio of N_2O_3 to N_2O_5 should be considerably greater than 1:1, while in all their experiments it was indeed somewhat smaller than 1. The absorption experiments with NaOH corresponded exactly with their earlier work; in absence of oxygen the results equalled those with H_2SO_4, in presence of free oxygen, oxidation took place in greater or less degree, according to the particular method of manipulation. Conclusion: "The repetition of experiments upon the behavior of N_2SO_4, with conc. H_2SO_4, on the one hand, and with NaOH on the other, taking the greatest possible care in regard to the purity of the substance, the apparatus and procedure, has proved that our previous results in this connection were perfectly correct, and that assertions of Raschig to the contrary must finally be disregarded as being absolutely wrong." II. Concerning the intermediate formation of **nitrogen trioxide** by the action of oxygen upon nitric oxide. In their earlier article they found that they had to offer new equations for the reactions in the lead chamber, based upon their experimental work, taking into consideration that on the one hand NO, on oxidation, yields directly nitrogen peroxide, without formation of the intermediate N_2O_3, and on the other hand that nitrosylsulphuric add is a carrier of oxygen, that is able to oxidize SO_2 to H_2SO_4 through the intermediate formation of the blue compound, sulphonitronic acid. They rejected the assertion of Raschig that the oxidation of NO to N_2O_4 takes place in two stages, the oxidation to N_2O_3 occurring very rapidly, that of N_2O_3 to N_2O_4 slowly, and that consequently a deflection must appear in the curve representing the reaction. They were able to show that a perfectly regular curve was obtained from their results and that the reaction $2NO+O_2=N_2O_4$ occurred regularly and without change of velocity, after presumably reaching the intermediate stage of N_2O_3. Protesting once more against Raschig's accusation of "intentional

distortion" of the curves, they draw two curves representing Raschig's results, "A," according to Raschig's own preference, giving the ratio between the nitrogen of the gases and the oxygen required for their oxidation-the ratio N: O=1, points to the formation of N_2O_3, that of N:O=2, to the formation of N_2O_4-"B" according to their own method based upon the percentage of the NO used which is converted into peroxide. No deflection being found in either "A" or "B," both Raschig's results graphically treated, and their own, therefore, confirm the view that the oxidation of NO proceeds directly to N_2O_4. The fact of their assuming 500 cc. air contained 125 cc. oxygen was an oversight, but did not affect the results, for it simply meant that instead of 100% excess of oxygen taken they used only 60% excess. They therefore recalculated the kinetics of the reaction according to Wegscheider's equation for the actual qantities taken, viz., 125 cc. NO and 500 cc. air, containing 100 cc. oxygen. Their earlier equation "D" for the reaction $2NO+O_2=2NO_2$, considering that the volumes at commencement Vo=6.25 times that of the oxygen used, becomes now: $dx/dt=K1\ (1.25-2x)2(1-x)/(6.25-x)2$ . . . (D1), and on integrating: $K1= 1/t\ (168.75\ /5-8x) + 112.25 \log (5-8x)-112.8 \log (1-x)\ -112.21)$. . . (Ia). As the results of the experiments. give the percentage of NO converted into N_2O_4, x, that is, the amount of the oxygen taken, used in the time t, is found as follows: %NO.times.5/8.times.100=0.00625 .times. $\%N_2O_4$. Their earlier equation "E," based upon the equation $2NO+O_2=0.5(2NO_2)+ 0.5\ N_2O_4$, becomes: $dx/dt=K2\ (1.25-2x)2(1-x)/(6.25-1.5x)2$ . . .(E1), and on integrating: $K2=1/t(150.5/(5-8x) + 91.06 \log (5-8x)-93.36 \log (1-x)-93.75)$ . . . (IIa). The results of the calculation of K1 and K2, according to equations Ia and IIa, are given in the tabulation: Duration, Composition of the gases on, "x", constants.;of, entering conc. H_2SO_4 in %, Amount of O;reaction, taken Used in, K1, K2;seconds., N_2O_4., NO., in time "t"., calc from Ia., calc. from IIa.;1.76, 52.49, 47-51, 0.3280, 11.63, 11.22;2.64, 61.33, 38.67, 0.3833, 11.71, 11.20;3.96, 69.05, 30.95, 0.4315, 11.56, 10.96;7.92, 80.56, 19.44, 0.5035, 11.91, 11.12;13.78, 85.28, 14.72, 0.5330, 10.11, 9.37;29.92, 91.77, 8.23, 0.5736, 9.92, 9.07 The conclusion arrived at from their earlier work that NO is oxidized directly to N_2O_4 must be maintained, on the ground of the graphic consideration, and on the basis of the recalculated results of the constants of the trimolecular reaction $2NO+O_2=N_2O_4$. Conclusion: That at ordinary temperatures NO is directly converted into N_2O_4, by free oxygen, without intermediate formation of N_2O_3, (**nitrous anhydride**) has been confirmed again and by methods which are free from objection. III. The Reactions in the Lead Chambers. Based upon the proved direct oxidation of NO to N_2O_4, the equations of the authors were formulated: (1) $SO_2+NO_2+H_2O=SO_5NH_2$ (sulphonitronic acid); (2a) $2SO_5.NH_2+O=H_2O+2SO_5NH$ (nitrosylsulphuric acid); (2b) $2SO_5NH_2+NO_2=HO+NO+2SO_5NH$; (3a) $2SO_5NH+H_2O=2H_2SO_4+NO+ NO_2$; (3b) $2SO_5NH+SO_2+2H_2O=H_2SO_4+2SO_5NH_2=$ (3c) $3SO_5NH_2=NO+H_2SO_4$: (4) $2NO+O_2=N_2O_4(2NO_2)$. Raschig denies (2a), (2a) and (2b), and says that (3b) does not exist, since SO_2 can react only with nitrous acid, forming sulphonitronic acid, and not with nitrosulphuric acid (nitrosulphonic acid). Against this Raschig states that Hg and Cu do not react with solutions of nitrosylsulphuric acid in H_2SO_4 of strengths below 80% and SO_2 does, while the authors considered the reaction of Hg, Cu and SO_2 as of the same order. Raschig endeavors to decide by the color reaction due to the formation of the blue-colored Cu salt of sulphonitronic acid, at what concentration of the H_2SO_4 the nitrous acid is free or combined in the form of nitrosulphonic acid. His positive statements are altogether in error; Weber, Trautz and the authors have shown that SO_2 can reduce nitrosylsulphuric add, even in conc. H_2SO_4, although more slowly than in the dilute acid, while, distinctly contrary to Raschig's statement, they have also shown that Cu and Hg reduce solutions of chamber crystals in

H_2SO_4, so dilute that certainly the major part Of the nitrogen is present as free HNO_2. For proof the authors dissolve nitrosylsulphuric acid in chamber acid of 52.degree. B.acte.e. The solution is of a yellowish green color and also shows by its odor that nitrosylsulphuric acid has been decomposed to form HNO_2 and H_2SO_4. By shaking the solution with Hg in a nitrometer, decomposition takes place with formation of pure NO and of Hg_2SO_4. That the blue color of the Cu salt of sulphonitronic acid cannot be detected in the presence of the dark gray mass, Hg_2SO_4, present in the nitrometer, when dil. H_2SO_4 is used, is a matter of course, but the formation of NO points to the same kind of reaction as in conc. H_2SO_4. That Cu can also reduce solutions of HNO_2 in dilute H_2SO_4 was shown by experiment. To prove reactions (1), (2a) and (2b), the authors take data from Raschig's own papers to establish their position. Raschig lets the red gases formed by action of oxygen on NO, and which, according to him, must consist of NO_2, pass into an aqueous solution of SO_2. The SO_2 not oxidized is boiled off, and the total acidity of the residual solution determined by titration. He then determined the H_2SO_4 present by precipitation with benzidine. According to Raschig, half the acidity found by NaOH should be ascribed to H_2SO_4, and the other half to HNO_3. This means that the following reactions take place: (1) $2N_2O_4+2H_2O=2HNO_3+2HNO_2$; (2) $2HNO_3+2HNO_2+ SO_2=2HNO_3+H_2SO_4+2NO$. According to Raschig, the HNO_3 is not then reduced by SO_2, but remains intact. But Raschig himself finds that 80% of the total acid is H_2SO_4 instead of the 50% which should be formed if the HNO_2 only is acted upon. The explanation is to be found in the fact that dilute HNO_3 while not reduced in water solution by SO_2 is easily and completely reduced in presence of H_2SO_4. In Raschig's experiment the concentration of the H_2SO_4 formed by the action of the HNO_2 was great enough to cause almost all of the HNO_2 to be reduced as well. Weber (Dingler's Pol. J., 181, 297, 1866) having called attention to this important point, the author repeated his experiments and found them correct. If a solution containing 1-2% of an 80% HNO_3 is made in H_2SO_4 of various strengths, when the density is 1.29 or above (1.72) the HNO_3 is reduced with extraordinary rapidity to pure NO. When SO_2 is ran in for sufficient length of time the reduction is complete, for in boiling off the SO_2 and testing it in the nitrometer, no NO is produced. The reaction takes place with especial rapidity with acids of the strength of chamber acids. Each gas bubble causes intense blue coloration, and the liquids become very warm. With sulphuric acid of about 1.72 containing HNO_3 the reaction goes on more slowly as previous observation had shown. But by titration with $KMnO_4$, after boiling off SO_2 it is shown that after a short time the passing of SO_2 reduces at least 11% of the HNO_3 to HNO_3, or nitrosylsulphuric acid. By still further action of SO_2 the solutions turn blue and NO is evolved, and if the liquid, after boiling, is tested in the nitrometer, so little NO is formed as to prove that even in acids of 60.degree. B.acte.e a reduction of HNO_3 to NO, through the intermediate step of sulphonitronic acid, takes place. This reaction occurring in the Glover tower when HNO_3 is added to make up loss of HNO_3, must take place with greater rapidity at the temperature there prevailing. Discussing their former equations: (1) $SO_2+NO_2+H_2O=SO_5NH_2$; (2b) $2SO_5NH_2+NO_2=NO+H_2O+2SO_5NH$. By doubling (1) and adding it to (2b) there results: (A) $2SO_2+3NO_2+2H_2O=NO+H_2O+2SO_5NH$. If this is true, one-third of the N present at first as NO_2 should appear as NO, while the rest would be in the compound SO_5NH, or free HNO_3. If then additional SO_2 reacts, the rest of the nitrogen shown in (A) as nitrosylsulphuric acid would be changed into NO. (B) $2SO_5NH+SO_2+2N_2O=2SO_5NH_2+H_2SO_4$(3b); $2SO_5NH_2=2H_2SO_4+2NO$ (equation (3c) doubled). (A) and (B) go on side by side. The reactions show that finally all the nitrogen is evolved as NO when the reactions take place in acid of the strength of chamber acid. The formulation of Raschig does not include

nitrosylsulphuric acid, only uses nitrous acid, and to the latter only ascribes the property of changing SO3 into H2SO4. The HNO3 formed from NO2 in his equations should remain unchanged. Therefore only half as much NO should be evolved in the end as would be expected from the formulation of the authors. To decide the point, a weighed amount of liquid NO2 is put into a gas evolution bottle, and from there driven by CO2 into a bottle containing 50 cc. chamber acid. Here it is met by SO2. An intense blue coloration of the acid at the point of union results and pure NO is evolved. There are difficulties in the quantitative determination, since NO in presence of NaOH (or of water) is changed by SO2 into N2O. (This may take place in the lead chamber in those regions where water is locally in excess) (PELOUZE, Ann. chim. phys., 60, 162; R. WEBER, Dingler's Pol. J., 184, 246, 1867: **LUNGE** AND BERL, Ber., 14, 2196; **LUNGE**, Soda Ind., Vol. 1, p. 635; and HEMPEL, Z. Elektrochem., 12, 600). They avoid this to some extent by condensing part of the SO2 in the gases by use of a freezing mixture. By collecting the NO thus freed from SO2 in a nitrometer over NaOH, 83% of the NO, according to (A) and (B), was obtained, and in another case 93%. In another experiment, weighed amounts of SO2 and NO2 were allowed to react. The amounts used were nearly in the ratio of 2SO2:3 NO2. There should then be an evolution of one-third of the nitrogen as NO (see equation (A)). The liquid SO2, however, evaporated more quickly than the N2O4, and further reduced some of the SO5NH formed, as indicated in (B). They find (as should be the case under this supposition) that more than one-third of the N appears as NO, viz., 45 cc. in place of 38.9 cc. Further they find that by leading in more SO2 a new quantity of NO is produced, as should be the case if nitrosylsulphuric acid, SO5NH or HNO2, is present (B). The new amount of NO is 55 cc., which, with the previous 45 cc., gives 100 cc., or about 85.5% of that to be expected from their theory. In this way they show the validity of equations (1), (2a) and (3b). The correctness of the equation (2a) is shown by their previous experiments (Z. angew. Chem., 19, 888). It has been shown that nitrosylsulphuric acid may act as an oxygen carrier, as expressed in (3b) and (2a). Their final conclusion is: The equations formerly advanced by us for the most important part of the chamber process ((1) SO2+NO2+H2O=SO5NH2 (sulphonitronic acid); (2b) 2SO5NH2+NO2=2SO5NH+H2O+NO; (3b) 2SO5NH+SO2+H2O=3H2SO4+2NO) have been newly investigated and not only established, but also found in sufficient approximation, quantitatively correct. It has also been proven: That the formation of nitrosyl-sulphuric acid and its decomposition with the intermediate step of sulphonitronic acid, plays the chief role in the chamber process. All of the opposing opinions of Raschig are incorrect; and further, also, the statement that HNO3 does not react with SO2 in the chamber process. Regarding the other equations under Section III, they say that (2a) has been shown on p. 838 of their former article to be certain; (3a) is, of course, not disputed by Raschig; (3c) was advanced by Raschig; and (4) receives in Section II proof in addition to that previously given (see C. A., 1907, 1896).

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=> d que l26
L21             1 SEA FILE=REGISTRY 10544-73-7
L22               SEL  L21 1- CHEM :      12 TERMS
L23          1279 SEA L22/BI
L25            13 SEA L23 AND (LUNG# OR PULMONARY)
L26            13 DUP REM L25 (0 DUPLICATES REMOVED)
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H2S

FILE 'REGISTRY' ENTERED AT 15:07:42 ON 29 JUN 2002
L30 1 S 7783-06-4

FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 15:08:06 ON 29 JUN 2002

FILE 'REGISTRY' ENTERED AT 15:08:19 ON 29 JUN 2002
SET SMARTSELECT ON
L31 SEL L30 1- CHEM : 12 TERMS
SET SMARTSELECT OFF

FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 15:08:20 ON 29 JUN 2002
L32 49751 S L31/BI
L33 4 S L32 AND HYPOXEMI?

L33 ANSWER 1 OF 4 MEDLINE
AN 2000496227 MEDLINE
DN 20307080 PubMed ID: 10850907
TI **Hydrogen sulfide** inhalation injury.
AU van Aalst J A; Isakov R; Polk J D; Van Antwerp A D; Yang M; Fratianne R B
CS Case Western Reserve University, MetroHealth Medical Center, Cleveland, Ohio, USA.
SO JOURNAL OF BURN CARE AND REHABILITATION, (2000 May-Jun) 21 (3) 248-53.
Journal code: 8110188. ISSN: 0273-8481.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Nursing Journals
EM 200010
ED Entered STN: 20001027
Last Updated on STN: 20001027
Entered Medline: 20001019
TI **Hydrogen sulfide** inhalation injury.
AB **Hydrogen sulfide** is a colorless, noxious gas with the distinctive smell of rotten eggs. This compound is a powerful reducing agent that is encountered in a number of industrial processes. When **hydrogen sulfide** is present, it exposes workers to the potentially lethal effects of the rapid **hypoxemia** that results from exposure to this agent. The "warning sign" is the characteristic smell of rotten eggs; this smell should alert anyone in the area that a potentially serious risk exists. The immediate removal of the victim and administration of high-flow oxygen is essential. Neurologic sequelae may require anticonvulsants and care must be exercised to observe for cardiac, hepatic, and renal insufficiency. Depending on the concentration, **hydrogen sulfide** can rapidly overcome a potential victim.
CT Check Tags: Case Report; Human; Male
Adult
Anoxemia
*Burns, Inhalation: CO, complications
*Burns, Inhalation: PA, pathology
***Hydrogen Sulfide: AE, adverse effects**
Inhalation Exposure
Middle Age
*Occupational Exposure
Oxygen: TU, therapeutic use
RN 7782-44-7 (Oxygen); **7783-06-4 (Hydrogen Sulfide)**

L33 ANSWER 2 OF 4 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2000359073 EMBASE
TI Distribution of hydrogen sulphide in rats' organs and associated histological changes in experimental intoxication.
AU Wachowiak R.; Tobolski J.; Miskowiak B.
CS R. Wachowiak, Department of Forensic Medicine, Medical Academy, Poznan, Poland
SO Z Zagadnien Nauk Sadowych, (2000) 43/- (275-282).
Refs: 12
ISSN: 1230-7483 CODEN: ZZSAF3
CY Poland
DT Journal; Conference Article
FS 005 General Pathology and Pathological Anatomy
049 Forensic Science Abstracts
052 Toxicology
LA English

SL English
CT Medical Descriptors:
*toxin analysis
*intoxication: DI, diagnosis
histopathology
organ distribution
hypoxemia: DI, diagnosis
autopsy
spectrophotometry
gas chromatography
thermal conductivity
nonhuman
rat
animal experiment
animal model
controlled study
animal tissue
conference paper
Drug Descriptors:
***hydrogen sulfide: TO, drug toxicity**
RN (**hydrogen sulfide**) 15035-72-0, **7783-06-4**

L33 ANSWER 3 OF 4 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2000214783 EMBASE
TI **Hydrogen sulfide** inhalation injury.
AU Van Aalst J.A.; Isakov R.; Polk J.D.; Van Antwerp A.D.; Yang M.; Fratianne R.B.
CS Dr. R.B. Fratianne, 2500 MetroHealth Dr, Cleveland, OH 44109-1998, United States
SO Journal of Burn Care and Rehabilitation, (2000) 21/3 (248-253).
Refs: 18
ISSN: 0273-8481 CODEN: JBCRD2
CY United States
DT Journal; Article
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
035 Occupational Health and Industrial Medicine
052 Toxicology
LA English
SL English
TI **Hydrogen sulfide** inhalation injury.
AB **Hydrogen sulfide** is a colorless, noxious gas with the distinctive smell of rotten eggs. This compound is a powerful reducing agent that is encountered in a number of industrial processes. When **hydrogen sulfide** is present, it exposes workers to the potentially lethal effects of the rapid **hypoxemia** that results from exposure to this agent. The 'warning sign' is the characteristic smell of rotten eggs; this smell should alert anyone in the area that a potentially serious risk exists. The immediate removal of the victim and administration of high-flow oxygen is essential. Neurologic sequelae may require anticonvulsants and care must be exercised to observe for cardiac, hepatic, and renal insufficiency. Depending on the concentration, **hydrogen sulfide** can rapidly overcome a potential victim.
CT Medical Descriptors:
*lung burn: ET, etiology
occupational exposure
lethality
hypoxemia: CO, complication
hypoxemia: TH, therapy

oxygen therapy
neurological complication: CO, complication
heart failure: CO, complication
liver failure: CO, complication
kidney failure: CO, complication
chemical industry
human
male
case report
adult
article
Drug Descriptors:
***hydrogen sulfide: TO, drug toxicity**
RN (**hydrogen sulfide**) 15035-72-0, **7783-06-4**

L33 ANSWER 4 OF 4 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 79010816 EMBASE
DN 1979010816
TI [Hazards of **hydrogen sulfide**].
GEFAHRDUNG DURCH SCHWEFELWASSERSTOFF - G11.
AU Becker B.
CS Enka AG, Oberbruch, Germany
SO Zentralblatt fur Arbeitsmedizin, Arbeitsschutz, Prophylaxe und Ergonomie, (1978) 28/8 (224-226).
CODEN: ZAAPDJ
CY Germany
DT Journal
FS 037 Drug Literature Index
035 Occupational Health and Industrial Medicine
017 Public Health, Social Medicine and Epidemiology
LA German
TI [Hazards of **hydrogen sulfide**].
GEFAHRDUNG DURCH SCHWEFELWASSERSTOFF - G11.
AB In acute poisoning, the clinical picture, with respiratory arrest, unconsciousness, convulsions and irritation of the upper respiratory tract, is unequivocal. The signs of chronic poisoning are those of **hypoxemia** and these can occur in any situation where there is lack of oxygen in the tissues as, for example, in CO poisoning. The precautions against acute poisoning are, and must remain, of a technical nature. In this connection, the physician can only ensure that the patient is healthy and fit for work. The prevention of chronic poisoning involves clinical tests of organs at risk for changes and disease resulting from H2S, whether it be the result of frequently repeated minor episodes of acute poisoning, or the questioned genuine chronic specific effect of H2S.
CT Medical Descriptors:
*health hazard
short survey
Drug Descriptors:
***hydrogen sulfide**
RN (**hydrogen sulfide**) 15035-72-0, **7783-06-4**

=>

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=> d his l39-

     (FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 15:19:11 ON 29 JUN 2002)

     FILE 'STNGUIDE' ENTERED AT 15:22:44 ON 29 JUN 2002

     FILE 'REGISTRY' ENTERED AT 15:32:01 ON 29 JUN 2002
L39            4 S 13826-64-7 OR 109-95-5 OR 13444-87-6 OR METHYL NITRITE/CN
L40            0 S PILOTY ACID/CN
L41            1 S PILOT? ACID
L42            5 S L39 OR L41

     FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 15:37:16 ON 29 JUN 2002

     FILE 'REGISTRY' ENTERED AT 15:37:30 ON 29 JUN 2002
                SET SMARTSELECT ON
L43          SEL L42 1- CHEM :      31 TERMS
                SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE, EMBASE' ENTERED AT 15:37:31 ON 29 JUN 2002
L44         37753 S L43/BI
L45           419 S (L44) AND (HYPOXEM? OR HYPOXIA OR ASTHMA? OR CYSTIC FIBRO? O
L46           287 DUP REM L45 (132 DUPLICATES REMOVED)
L47            55 S L46 AND HYPOXEM?

=> d que l47
L39            4 SEA FILE=REGISTRY 13826-64-7 OR 109-95-5 OR 13444-87-6 OR
                  METHYL NITRITE/CN
L41            1 SEA FILE=REGISTRY PILOT? ACID
L42            5 SEA FILE=REGISTRY L39 OR L41
L43              SEL  L42 1- CHEM :      31 TERMS
L44         37753 SEA L43/BI
L45           419 SEA (L44) AND (HYPOXEM? OR HYPOXIA OR ASTHMA? OR CYSTIC FIBRO?
                  OR ARD OR ADULT RESPIRATORY DISTRESS OR PNEUMON? OR INTERSTITIA
                  L LUNG DISEASE#)
L46           287 DUP REM L45 (132 DUPLICATES REMOVED)
L47            55 SEA L46 AND HYPOXEM?


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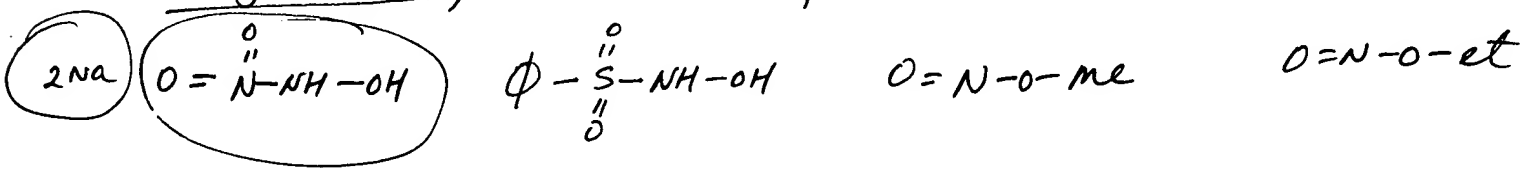
All hits have one of 5 Compounds + Hypoxem?

Angeli's salt, Piloty's Acid, methyl nitrite, ethyl nitrite



Nitrosyl Bromide

L47 ANSWER 1 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 2001:361442 CAPLUS
DN 135:190167
TI S-nitrosothiol repletion by an inhaled gas regulates pulmonary function
AU Moya, Martin P.; Gow, Andrew J.; McMahon, Timothy J.; Toone, Eric J.; Cheifetz, Ira M.; Goldberg, Ronald N.; Stamler, Jonathan S.
CS Neonatal-Perinatal Research Institute, Department of Pediatrics, Duke University Medical Center, Durham, NC, 27710, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2001), 98(10), 5792-5797
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB NO synthases are widely distributed in the lung and are extensively involved in the control of airway and vascular homeostasis. It is recognized, however, that the O2-rich environment of the lung may predispose NO toward toxicity. These Janus faces of NO are manifest in recent clin. trials with inhaled NO gas, which has shown therapeutic benefit in some patient populations but increased morbidity in others. In the airways and circulation of humans, most NO bioactivity is packaged in the form of S-nitrosothiols (SNOs), which are relatively resistant to toxic reactions with O2/O2-. This finding has led to the proposition that channeling of NO into SNOs may provide a natural defense against lung toxicity. The means to selectively manipulate the SNO pool, however, has not been previously possible. Here we report on a gas, O-nitrosoethanol (ENO), which does not react with O2 or release NO and which markedly increases the concn. of indigenous species of SNO within airway lining fluid. Inhalation of ENO provided immediate relief from hypoxic pulmonary vasoconstriction without affecting systemic hemodynamics. Further, in a porcine model of lung injury, there was no rebound in cardiopulmonary hemodynamics or fall in oxygenation on stopping the drug (as seen with NO gas), and addnl. ENO protected against a decline in cardiac output. Our data suggest that SNOs within the lung serve in matching ventilation to perfusion, and can be manipulated for therapeutic gain. Thus, ENO may be of particular benefit to patients with pulmonary hypertension, **hypoxemia**, and/or right heart failure, and may offer a new therapeutic approach in disorders such as **asthma** and **cystic fibrosis**, where the airways may be depleted of SNOs.
IT Cardiovascular system
Circulation
Hypoxia, animal
Lung
Vasoconstriction
(S-nitrosothiol repletion by O-nitrosoethanol regulates pulmonary function)
IT **109-95-5**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(S-nitrosothiol repletion by O-nitrosoethanol regulates pulmonary function)

L47 ANSWER 2 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 2001:185620 CAPLUS
DN 134:202701

TI Method of treating cardiopulmonary diseases with NO group compounds
IN Stamler, Jonathan S.; Toone, Eric J.; Gow, Andrew J.
PA Duke University, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001017596	A1	20010315	WO 2000-US20784	20000818
	W: AU, CA, JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 6314956	B1	20011113	US 1999-390215	19990908
PRAI	US 1999-390215	A	19990908		

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Treatment of pulmonary disorders assocd. with **hypoxemia** and/or smooth muscle constriction and/or inflammation comprises administering into the lungs as a gas a compd. with an NO group which does not form NO2/NOx in the presence of oxygen or reactive oxygen species at body temp. Treatment of cardiac and blood disorders, e.g., angina, myocardial infarction, heart failure, hypertension, sickle cell disease and clotting disorders, comprises administering into the lungs as a gas, a compd. which reacts with cysteine in Hb and/or dissolves in blood and has an NO group which is bound in the compd. so that it does not form NO2/NOx in the presence of oxygen or reactive oxygen species at body temp. Exemplary of the compd. administered in each case is Et nitrite. Treatment of patient in need of improved oxygenation, blood flow of and/or thinning of blood comprises providing in the patient a therapeutic amt. of red blood cells loaded with nitrosylated Hb. A method is also provided for screening drugs that increase level of nitrosoglutathione in airway lining fluid.

ST NO compd cardiopulmonary disease treatment; **ethyl nitrite** cardiopulmonary disease treatment; nitrosylated Hb cardiopulmonary disease; airway lining fluid nitrosoglutathione modulator screening

IT Anti-inflammatory agents
Anti-ischemic agents
Antiasthmatics
Anticoagulants
Antihypertensives
Cardiovascular agents
Cystic fibrosis
Drug screening
Erythrocyte
Hypoxia, animal
Lung, disease
Sickle cell anemia
Vasodilators
(NO group compds. for treating cardiopulmonary diseases)

IT **Hypoxia**, animal
(**hypoxemia**; NO group compds. for treating cardiopulmonary diseases)

IT **109-95-5, Ethyl nitrite** 616-91-1, N-Acetylcysteine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(NO group compds. for treating cardiopulmonary diseases)

L47 ANSWER 3 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 2000:326014 CAPLUS
DN 133:236333
TI Interactive effects of anoxia and general anesthesia during birth on the degree of CNS and systemic **hypoxia** produced in neonatal rats
AU Berger, Neil; Vaillancourt, Cathy; Boksa, Patricia
CS Department of Psychiatry, Douglas Hospital Research Centre, McGill University, Verdun, QC, H4H 1R3, Can.
SO Experimental Brain Research (2000), 131(4), 524-531
CODEN: EXBRAP; ISSN: 0014-4819
PB Springer-Verlag
DT Journal
LA English
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Interactive effects of anoxia and general anesthesia during birth on the degree of CNS and systemic **hypoxia** produced in neonatal rats
AB A model of global **hypoxia** during Caesarean-section (C-section) birth was widely used to study long-term effects of birth **hypoxia** on central nervous system (CNS) function. However, the actual degree of CNS and systemic **hypoxia** produced by the birth insult in this model has never been characterized. Addnl., the way in which the dam is anesthetized during the C-section procedure may impinge on the degree of **hypoxia** experienced by the neonate. This study examd. how a period of global birth anoxia and isoflurane/**N2O** anesthesia interact to affect measures of CNS and systemic **hypoxia** in neonatal rats born by C-section compared with control, vaginally born animals. A 10-min period of global anoxia just before birth increased blood lactate, a metabolic indicator of systemic **hypoxia**, increased brain lactate and decreased brain ATP to a similar extent in pups born by C-section from either decapitated, unanaesthetised dams or dams anesthetized with 2.5% isoflurane. Thus, this model does produce systemic and CNS **hypoxia** in the neonate. Pups born by C-section with a higher concn. of isoflurane (3.5%), in the absence of added global anoxia, also showed redns. in brain ATP at birth. In addn., 10 min of global anoxia produced greater increases in blood lactate in pups born from dams anesthetized with the higher concn. of isoflurane. Thus, the concn. of anesthetic used in this model may affect the degree of CNS or systemic **hypoxia** experienced by the neonate. Compared with vaginal birth, pups born by C-section with 2.5% or 3.5% isoflurane (and no added global anoxia) showed decreased pO2 and pH, and increased pCO2 in systemic blood taken <30 s after birth. Exposure to global anoxia during C-section birth actually increased systemic pO2 at <30 s after birth, presumably due to ventilatory responses to **hypoxemia** and hypercapnia; this effect of anoxia was reduced in anesthetized compared with unanaesthetised pups. Thus, global anoxia acts as a stimulus for rapid recovery of systemic pO2 at birth, and this stimulus is dampened by isoflurane/**N2O** anesthesia. These results should aid in understanding how CNS and systemic **hypoxia** at birth contribute to long-term changes in brain biochem. and behavior in this model.
ST newborn **hypoxia** central nervous system metab; lactate blood brain **hypoxia** neonate; ATP brain **hypoxia** neonate; carbon dioxide blood brain **hypoxia** neonate; oxygen blood brain **hypoxia** neonate
IT **Hypoxia**, animal
Newborn
(anoxia and general anesthesia during birth effect on the degree of CNS

and systemic **hypoxia** in neonatal)

IT Nervous system
(central; anoxia and general anesthesia during birth effect on the degree of CNS and systemic **hypoxia** in neonatal)

L47 ANSWER 4 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 2000:109450 CAPLUS
DN 133:533
TI Inhaled nitric oxide delivery by anesthesia machines
AU Ceccarelli, Patrizia; Bigatello, Luca M.; Hess, Dean; Kwo, Jean; Melendez, Luis
CS Department of Anesthesia and Critical Care and the Respiratory Care Services, Harvard Medical School, Massachusetts General Hospital, Boston, MA, 02114, USA
SO Anesthesia & Analgesia (Baltimore) (2000), 90(2), 482-488
CODEN: AACRAT; ISSN: 0003-2999
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Inhaled nitric oxide (NO) is a selective pulmonary vasodilator used to treat intraoperative pulmonary hypertension and **hypoxemia**. In contrast to NO delivered by crit. care ventilators, NO delivered by anesthesia machines can be complicated by rebreathing. We evaluated two methods of administering NO intraoperatively; via the nitrous oxide (**N2O**) flowmeter and via the INOvent (Datex-Ohmeda, Madison, WI). We hypothesized that both systems would deliver NO accurately when the fresh gas flow (FGF) rate was higher than the minute ventilation (VE). Each system was set to deliver NO to a lung model. Rebreathing of NO was obtained by decreasing FGF and by simulating partial NO uptake by the lung. At FGF .gtoreq. VE (6 L/min), both systems delivered an inspired NO concn. ([NO]) within approx. 10% of the [NO] set. At FGF < VE and complete NO uptake, the **N2O** flowmeter delivered a lower [NO] (70 and 40% of the [NO] set at 4 and 2 L/min, resp.) and the INOvent delivered a higher [NO] (10 and 23% higher than the [NO] set at 4 and 2 L/min, resp.). Decreasing the NO uptake increased the inspired [NO] similarly with both systems. At 4 L/min FGF, [NO] increased by 10%-20% with 60% uptake and by 18%-23% with 30% uptake. At 2 L/min, [NO] increased by 30%-33% with 60% uptake and by 60%-69% with 30% uptake. We conclude that intraoperative NO inhalation is accurate when administered either by the **N2O** flowmeter of an anesthesia machine or by the INOvent when FGF .gtoreq. VE. Inhaled nitric oxide (NO) is a selective pulmonary vasodilator. In a lung model, we demonstrated that NO can be delivered accurately by a **N2O** flowmeter or by a com. device. We provide guidelines for intraoperative NO delivery.

L47 ANSWER 5 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1998:769893 CAPLUS
DN 130:177420
TI Respiration during emergence from anesthesia with desflurane/**N2O** vs. desflurane/air for gynecological laparoscopy
AU Einarsson, S. G.; Cerne, A.; Bengtsson, A.; Stenqvist, O.; Bengtson, J. P.
CS Departments of Anaesthesia and Intensive Care, Sahlgrenska University Hospital, University of Goteborg, Swed.
SO Acta Anaesthesiologica Scandinavica (1998), 42(10), 1192-1198
CODEN: AANEAB; ISSN: 0001-5172
PB Munksgaard International Publishers Ltd.
DT Journal

LA English
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Respiration during emergence from anesthesia with desflurane/**N2O** vs. desflurane/air for gynecological laparoscopy
AB The complications related to anesthesia usually occur in the early postoperative period. Hypercapnia and **hypoxemia** may result from any persistent depression of the respiratory drive relative to the metabolic demand. The purpose of this study was to compare the respiratory effects of desflurane anesthesia with or without nitrous oxide during the period of emergence. Twenty patients scheduled for a standardized surgical procedure, laparoscopic hysterectomy, were randomly allocated to anesthesia with 1.3 MAC of desflurane/**N2O** (Group 1) or desflurane alone (Group 2), with 10 patients in each group. Times of resumption of spontaneous breathing and extubation were recorded and elimination rates of carbon dioxide, end-tidal concns. of desflurane and **N2O**, and blood gases were measured. Spontaneous breathing was resumed in both groups when pH had decreased by about 0.07 and PaCO2 increased by about 1.4 kPa compared with the values at the end of 1.3 MAC anesthesia with controlled normoventilation. There were no significant differences between the groups with regards to extubation time, 6 vs. 13 min, or total MAC value at extubation, 0.20 vs. 0.19 in Group 1 and 2, resp. Neither did the groups differ in minute ventilation, end-tidal carbon dioxide, oxygen concns., or blood gases. CO2 elimination decreased in both groups from about 220 mL 70kg-1 min-1 at the end of anesthesia to a lowest value of about 160 mL 70 kg-1 min-1. The respiratory profiles during recovery from gynaecol. laparoscopy with either desflurane/**N2O** or desflurane anesthesia were similar with fast resumption of spontaneous breathing, short time to extubation, and no signs of CO2 retention.
ST desflurane **N2O** respiration anesthesia laparoscopy
IT Anesthetics
Breathing (animal)
(desflurane/**N2O** vs. desflurane/air effects on respiration during emergence from anesthesia for gynecol. laparoscopy in humans)
IT Abdomen
Abdomen
Surgery
Surgery
(laparoscopy; desflurane/**N2O** vs. desflurane/air effects on respiration during emergence from anesthesia for gynecol. laparoscopy in humans)
IT 10024-97-2, Nitrogen oxide (**N2O**), biological studies
57041-67-5, Desflurane
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(desflurane/**N2O** vs. desflurane/air effects on respiration during emergence from anesthesia for gynecol. laparoscopy in humans)
IT 124-38-9, Carbon dioxide, biological studies 7782-44-7, Oxygen, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(desflurane/**N2O** vs. desflurane/air effects on respiration during emergence from anesthesia for gynecol. laparoscopy in humans)

L47 ANSWER 6 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1998:624130 CAPLUS
DN 129:229145
TI Cardiovascular function and brain metabolites in normal weight and

intrauterine growth restricted newborn piglets. Effect of mild **hypoxia**

AU Bauer, Reinhard; Walter, Bernd; Glaser, Elke; Roesel, Thomas; Kluge, Harald; Zwiener, Ulrich

CS Inst. Pathophysiology, Friedrich Schiller Univ., Jena, D-07740, Germany

SO Experimental and Toxicologic Pathology (1998), 50(4-6), 294-300
CODEN: ETPAEK; ISSN: 0940-2993

PB Gustav Fischer Verlag

DT Journal

LA English

TI Cardiovascular function and brain metabolites in normal weight and intrauterine growth restricted newborn piglets. Effect of mild **hypoxia**

AB Newborns were divided in normal wt. (NW, birth wt. > 40th percentile) and intrauterine growth restricted (IUGR, birth wt. > 5th and < 10th percentiles) piglets and were anesthetized with halothane in 70% **N2O** and 30% O2, and after immobilization artificially ventilated. The acid-base balance and blood gas values at baseline conditions were similar within the different groups and consistent with other data obtained from anesthetized and artificially ventilated newborn piglets. Mild hypoxic **hypoxia** which was induced by lowering the inspired fraction of O2 (FiO2) from 0.35 to 0.15 resulted in reduced arterial pO2 (NW: from 115 to 39, IUGR: from 117 mm Hg to 39 mm Hg), but arterial pH and pCO2 remained unchanged. Under baseline conditions arterial blood pressure, cardiac output, and myocardial contractility (dp/dtmax), and blood plasma catecholamine values were similar in all groups. Heart rate was slightly increased in IUGR. Mild **hypoxia** led to a strong increase of myocardial contractility in NW as well as IUGR piglets to 2.4-2.7-fold and remained increased during recovery. Total peripheral resistance was enhanced at the end of recovery period in IUGR animals. There was an increase of epinephrine (E) in NW compared to sham-operated animals. During reoxygenation the further increase in E and norepinephrine levels were enhanced in the animals which suffered from mild **hypoxia**. Regional distribution of brain tissue metabolites was partly affected by intrauterine growth restriction. Brain tissue Glc content was strongly reduced by 65-72% in all brain regions investigated. Mild **hypoxia** led to an increase of 30% in NW animals. The percentage increase of brain Glc content in IUGR piglets was more pronounced but with higher variance. A strong increase of brain lactate content appeared here. Brain tissue ATP was quite similar in all groups, but brain creatine phosphate was reduced in some forebrain structures of IUGR piglet after mild **hypoxia**. Mild **hypoxemia** was well tolerated of both groups. Lactate was supposed to play a significant role as a source for brain energy prodn. in the newborn IUGR piglets.

ST **hypoxia** cardiovascular function brain metabolite piglet

IT Blood pressure
Brain
Cardiovascular system
Heart rate
Hypoxia, animal
Newborn
Pregnancy
(effect of mild **hypoxia** on cardiovascular function and brain metabolites in normal wt. and intrauterine growth restricted newborn piglets)

IT Embryo, animal
(fetus; effect of mild **hypoxia** on cardiovascular function and brain metabolites in normal wt. and intrauterine growth restricted newborn piglets)

IT 50-21-5, Lactic acid, biological studies 50-99-7, Glucose, biological studies 56-65-5, ATP, biological studies 67-07-2, Creatine phosphate
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(brain tissue; effect of mild **hypoxia** on cardiovascular function and brain metabolites in normal wt. and intrauterine growth restricted newborn piglets)

IT 51-41-2, Norepinephrine 51-43-4, Epinephrine 51-61-6, Dopamine, biological studies 124-38-9, Carbon dioxide, biological studies 7782-44-7, Oxygen, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(effect of mild **hypoxia** on cardiovascular function and brain metabolites in normal wt. and intrauterine growth restricted newborn piglets)

L47 ANSWER 7 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1997:809346 CAPLUS
DN 128:123741
TI Emergence from isoflurane/**N2O** or isoflurane anesthesia
AU Einarsson, S.; Bengtsson, A.; Stenqvist, O.; Bengtson, J. P.
CS Department of Anaesthesia and Intensive Care, Sahlgrenska University Hospital, University of Goteborg, Goteborg, Swed.
SO Acta Anaesthesiologica Scandinavica (1997), 41(10), 1292-1299
CODEN: AANEAB; ISSN: 0001-5172
PB Munksgaard International Publishers Ltd.
DT Journal
LA English
TI Emergence from isoflurane/**N2O** or isoflurane anesthesia
AB The first goal of anesthetic recovery is return of the patient's ability to independently maintain respiratory and circulatory functions. Nitrous oxide remains popular due to minor effects on the cardiovascular and respiratory systems. However, diffusion **hypoxemia** can occur during recovery and there is a potential advantage of providing the patient with only a potent vaporized agent. This randomized study of 20 gynecol. patients evaluated respiratory and circulatory variables during emergence after anesthesia with equipotent mixts. of isoflurane/nitrous oxide or isoflurane. Inspired, end-tidal and mixed expired gas concns., expired minute vol., pulse oximetry satn. and arterial blood gases were registered. Monitoring of cardiac output was performed by transthoracic bioimpedance. Patients anesthetized with isoflurane/**N2O** resumed their spontaneous breathing 16 min earlier and were extubated 22 min earlier than those anesthetized with only isoflurane. At extubation, total MAC and end-tidal CO2 were similar in both groups, 0.22-0.26 and 5.5-5.9 vol%, resp. The isoflurane / **N2O** group had greater minute ventilation and CO2 excretion rates than the isoflurane group throughout the emergence period. There were no significant differences between the groups in blood gas variables or in heart rate, mean arterial blood pressure or cardiac index. Cardiac index was between 3.4 and 3.9 l m-2 min-1 throughout the emergence period in both groups. Patients anesthetized with only isoflurane had a longer delay until resumption of spontaneous breathing and extubation in the emergence period. Minute ventilation and carbon dioxide elimination were also significantly more suppressed throughout emergence after anesthesia with isoflurane as compared with isoflurane/**N2O**.
IT Anesthetics
Circulation
Hypoxia, animal
Respiration, animal

(isoflurane/**N2O** vs. isoflurane anesthetic recovery in humans)

IT 10024-97-2, Nitrous oxide, biological studies 26675-46-7, Isoflurane
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(isoflurane/**N2O** vs. isoflurane anesthetic recovery in humans)

L47 ANSWER 8 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1997:809345 CAPLUS
DN 128:123740
TI Should nitrous oxide be discontinued before desflurane after anesthesia with desflurane/**N2O**?
AU Einarsson, S.; Cerne, A.; Bengtsson, A.; Stenqvist, O.; Bengtson, J. P.
CS Department of Anaesthesiology, Sahlgrenska University Hospital, University of Goteborg, Goteborg, Swed.
SO Acta Anaesthesiologica Scandinavica (1997), 41(10), 1285-1291
CODEN: AANEAB; ISSN: 0001-5172
PB Munksgaard International Publishers Ltd.
DT Journal
LA English
TI Should nitrous oxide be discontinued before desflurane after anesthesia with desflurane/**N2O**?
AB The appearance of **hypoxemia** immediately after anesthesia with nitrous oxide may be partially explained by diffusion **hypoxia**. This study was undertaken to evaluate circulatory and respiratory variables during emergence after desflurane/nitrous oxide anesthesia, and whether there are any differences depending on which gas is discontinued first. 20 Patients were studied after gynaecol. laparoscopic surgery. The depth of anesthesia was reduced 10 min prior to the emergence by stopping the administration of one of the two inhalational agents. Desflurane was discontinued first in Group 1, nitrous oxide in Group 2. Ventilation was controlled with E'CO2 maintained at 5% until the administration of the second anesthetic gas was discontinued. Thereafter, the patients breathed spontaneously. The PaCO2 at which the respiratory drive reappeared after controlled normoventilation was similar in both groups, 6.1-6.5 kPa, and extubation was performed after 10-11 min. At extubation, the end-tidal CO2 and total MAC were similar in the groups, about 6.2 vol% and 0.16, resp. Mean arterial blood pressure was significantly higher in Group 1. The cardiac output increased in both groups from about 6 l/min at the conclusion of anesthesia to 9.0 and 7.6 l/min at 15 min in the recovery period. End-tidal O2 decreased and CO2 increased in both groups during the first 10 min in the recovery period. PH was reduced at 15 and 30 min in both groups. Irresp. of which agent was discontinued first, there was an increase in cardiac output, decrease in oxygenation and a modest acidosis in the first 30-min recovery period. The only significant difference between the groups was in mean arterial blood pressure in the early emergence phase with a greater MAP when **N2O** had been used until the conclusion of anesthesia.
IT Anesthetics
Blood pressure
Circulation
Hypoxia, animal
Respiration, animal
(desflurane/**N2O** discontinuation sequence effects on postoperative recovery in humans)
IT 10024-97-2, Nitrous oxide, biological studies 57041-67-5, Desflurane
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(desflurane/**N2O** discontinuation sequence effects on postoperative recovery in humans)

L47 ANSWER 9 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1993:16195 CAPLUS
DN 118:16195
TI The effect of a 50% inspired mixture of nitrous oxide on arterial oxygen tension in spontaneously breathing horses anesthetized with halothane
AU Young, L. E.; Richards, D. L. S.; Brearley, J. C.; Bartram, D. H.; Jones, R. S.
CS Univ. Dep. Anaesth., R. Liverpool Hosp., Liverpool, L63 3BX, UK
SO J. Vet. Anaesth. (1992), 19, 37-40
CODEN: JVANEJ
DT Journal
LA English
AB Administration of 50% **N2O** decreased arterial pO2 in halothane-anesthetized horses in lateral and dorsal recumbency; however, when administered to horses in lateral recumbency it did not promote arterial **hypoxemia**. There was a higher risk of intraoperative arterial **hypoxemia** assocd. with its use in spontaneously breathing horses in dorsal recumbency. Arterial **hypoxemia** occurred in all the horses during the 1st 15 min of recovery, but when **N2O** was discontinued, supplying halothane in O to the breathing circuit for 5 min at a flow rate of 20 mL/kg/min was sufficient to ensure that diffusion **hypoxia** did not occur. The time taken to achieve sternal recumbency was shorter in the horses that had received **N2O** than in those that had not.
ST horse anesthesia halothane nitrous oxide **hypoxemia**; oxygen blood halothane anesthesia nitrous oxide

L47 ANSWER 10 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1990:19570 CAPLUS
DN 112:19570
TI Conditions for pharmacologic evaluation in the gerbil model of forebrain ischemia
AU Clifton, Guy L.; Taft, William C.; Blair, Robert E.; Choi, Sung C.; DeLorenzo, Robert J.
CS Med. Coll. Virginia, Virginia Commonw. Univ., Richmond, VA, USA
SO Stroke (Dallas) (1989), 20(11), 1545-52
CODEN: SJCCA7; ISSN: 0039-2499
DT Journal
LA English
AB Inspired oxygen concn. (FiO2), choice of anesthetic, nutritional status, and body temp. were examd. in a gerbil model of forebrain ischemia to det. their effect on data interpretation, ischemic outcome, and extent of pharmacol. protection. Four hundred eight-four gerbils were subjected to 5 min of forebrain ischemia under different exptl. conditions. The gerbils were anesthetized with 3% halothane and inspired 21% O2, 37% O2 and 60% **N2O**, or 97% O2. Six groups of gerbils pretreated with 200 mg/kg phenytoin or 2 mL/kg polyethylene glycol (vehicle) underwent ischemia in the fasted or fed state. Three groups of gerbils receiving no pretreatment underwent ischemia with rectal temps. of 32-33.degree., 34-35, or 37.degree.. The authors counted intact neurons in the CA1 hippocampal sector in brains fixed on Day 7 after ischemia, and t tests of square-root-transformed cell counts were used to assess the effect of hypothermia, and anal. of variance of the transformed data was used to test for the effects of phenytoin, FiO2, and nutritional status. Phenytoin pretreatment provided significant protection from CA1 neuron loss in all groups tested, but the degree of protection varied from 20% to

44%. In spite of higher serum glucose concns. in fed than in fasted gerbils, no significant effect of nutritional status upon neuron loss in phenytoin- or vehicle-pretreated gerbils was found. An FiO2 of 21% decreased the no. of viable neurons in both vehicle- and phenytoin-pretreated groups when compared with greater FiO2s, despite the lack of an effect of **hypoxemia** on arterial blood gases. Body temp. during ischemia had a dramatic impact on ischemia-induced cell death. Even 2.degree. of hypothermia provided 100% protection from cerebral ischemia. Thus, a min. of 20 gerbils per group together with rigorous attention to detail are necessary to reliably det. protective effect and therapeutic efficacy in this widely used model.

L47 ANSWER 11 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1989:567028 CAPLUS
DN 111:167028
TI The effect of etomidate pretreatment on cerebral high energy metabolites, lactate, and glucose during severe **hypoxia** in the rat
AU Smith, David S.; Keykhah, M. Mehdi; O'Neill, John J.; Harp, James R.
CS Dep. Anesthesiol., Temple Univ., Philadelphia, PA, USA
SO Anesthesiology (1989), 71(3), 438-43
CODEN: ANESAV; ISSN: 0003-3022
DT Journal
LA English
TI The effect of etomidate pretreatment on cerebral high energy metabolites, lactate, and glucose during severe **hypoxia** in the rat
AB Etomidate was compared with thiopental with respect to preventing loss of brain high-energy metabolites and accumulation of lactate during 20 min of **hypoxemia** (PaO2 of 16-19 mmHg) in rats with unilateral carotid artery ligation. Male rats anesthetized with halothane and **N2O** in O were randomly assigned to 6 groups. The normoxic control group received 70% **N2O** in O, the hypoxic group received no i.v. drug treatment (**hypoxia-N2O**), and 4 i.v. drug treatment groups (**N2O** was replaced by 70% N at the start of drug administration). The i.v. drug groups were treated with **hypoxia**-etomidate low dose (1 mg/kg followed by an infusion at 0.35 mg/kg/min); **hypoxia**-etomidate high dose (1 mg/kg, then 1.3 mg/kg/min); **hypoxia**-thiopental low dose (15 mg/kg, then 1.5 mg/kg/min); and **hypoxia**-thiopental high dose (15 mg/kg, then 5 mg/kg/min). Brain metabolite concns. on the side ipsilateral to the ligated carotid artery in the normoxia-**N2O** group were ATP 2.76, phosphocreatine (PCr) 3.88, lactate 2.34, and glucose 3.56 (.mu.mol/g wet wt.). There was no decrease in ATP in any of the hypoxic groups. PCr decreased by 45% (compared to normoxia-**N2O**) in the **hypoxia-N2O** group. In the i.v. drug treatment groups, only the **hypoxia**-thiopental high dose group had decreased PCr. Lactate increased in all hypoxic groups, though it was highest in the **hypoxia-N2O** group (24.3 .mu.mol/g). Brain glucose did not change as a function of the drug treatment. In this model, both high- and low-dose etomidate and low-dose thiopental prevented the decrease in PCr that the decrease occurred when **N2O** alone was used. Etomidate and thiopental also attenuated, but did not prevent the increase in brain lactate. Thus, etomidate may prevent metabolic changes and cell damage during **hypoxemia**.
ST brain **hypoxia** etomidate thiopental
IT **Hypoxia**
(brain high-energy metabolites response to, etomidate effect on)
IT Brain, disease or disorder
(ischemia, high-energy metabolites response to **hypoxia** in, etomidate effect on)

IT 76-75-5, Thiopental 33125-97-2, Etomidate
RL: BIOL (Biological study)
(brain high-energy metabolites response to, in **hypoxia**)
IT 50-21-5, Lactic acid, biological studies 50-99-7, Glucose, biological studies 56-65-5, 5'-ATP, biological studies 67-07-2, Phosphocreatine
RL: BIOL (Biological study)
(of brain in **hypoxia**, etomidate or thiopental effect on)

L47 ANSWER 12 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1983:464258 CAPLUS
DN 99:64258
TI Cerebral protection by isoflurane during **hypoxemia** or ischemia
AU Newberg, L. A.; Michenfelder, J. D.
CS Dep. Anesthesiol., Mayo Clin., Rochester, MN, 55905, USA
SO Anesthesiology (1983), 59(1), 29-35
CODEN: ANESAV; ISSN: 0003-3022
DT Journal
LA English
TI Cerebral protection by isoflurane during **hypoxemia** or ischemia
AB The possible cerebral protective effects of isoflurane [26675-46-7] against **hypoxemia** and ischemia were studied in mice and dogs, resp. In mice breathing 5% O, survival time was increased significantly over controls in groups exposed to 1.0% and 1.4% isoflurane. At higher concns. (2.0% and 3.0%) it is presumed that cardiorespiratory depression contributed to shorter survival times. In dogs, the effects of 3% isoflurane on the rates of cerebral ATP and phosphocreatine depletion and lactate accumulation during incomplete global ischemia were compared with control dogs exposed to **N2O**. Incomplete global ischemia was produced by acute hemorrhagic hypotension (30 mmHg for 9 min), a situation that does not abolish cortical elec. activity (active EEG). In the dogs exposed to isoflurane, the cerebral energy stores of ATP and phosphocreatine and the cerebral energy charge were sustained at significantly higher levels than in dogs exposed to **N2O**, and the cerebral lactate accumulation was significantly less in the initial 7 min of hypotension. Thus, in circumstances of O deprivation insufficient to abolish cortical elec. activity, isoflurane, like the barbiturates, can provide some cerebral protection, presumably by depressing cortical elec. activity and cerebral metab.
ST isoflurane brain protection **hypoxemia** ischemia
IT Brain, metabolism
(isoflurane effect on, during **hypoxemia** and ischemia)
IT 26675-46-7
RL: BIOL (Biological study)
(brain protection by, during **hypoxemia** and ischemia, brain metab. and elec. activity in relation to)

L47 ANSWER 13 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1983:213365 CAPLUS
DN 98:213365
TI Carbohydrate and energy metabolism of the aging rat brain in severe arterial **hypoxemia**
AU Degrell, Istvan; Krier, Claude; Hoyer, Siegfried
CS Dep. Pathochem. Gen. Neurochem., Univ. Heidelberg, Heidelberg, 6900, Fed. Rep. Ger.
SO Aging (N. Y.) (1983), 21, 289-300
CODEN: AGNYDE; ISSN: 0160-2721
DT Journal
LA English
TI Carbohydrate and energy metabolism of the aging rat brain in severe

arterial **hypoxemia**

AB Brain carbohydrate and energy metab. were studied to investigate the effect of severe arterial **hypoxemia** in 1- and 2-yr-old rats. Twenty control and 10 **hypoxemia** rats in each age group were anesthetized with **N2O** and halothane, immobilized, and artificially ventilated. After a 15-min steady state of arterial normotension, normocapnia and normoxemia, paO2 was kept normoxemic in the controls and lowered to approx. 21 mm Hg in the **hypoxemic** groups for a further 15 min. Under the steady-state conditions mentioned, the brains were frozen in situ with liq. N. The brain cortex was analyzed for the concns. of glucose, glucose 6-phosphate (G-6-P), fructose 6-phosphate (F-6-P), fructose 1,6-diphosphate (F-1,6-P), dihydroxyacetone phosphate (DHAP), pyruvate, lactate, citrate, .alpha.-ketoglutarate, malate, creatine phosphate, ATP, ADP, and AMP, using sensitive enzymic methods. In the control groups there was no significant difference between the concns. of metabolites in 1- and 2-yr-old rats. Severe arterial **hypoxemia** in 1-yr-old rats increases the concns. of glucose, G-6-P, F-1,6-P, DHAP, pyruvate, and lactate, indicating an increased activity of the flux-controlling glycolytic enzymes hexokinase, phosphofructokinase, and pyruvate kinase. The concns. of creatine phosphate and ATP were lowered, and the levels of ADP and AMP were elevated, but the adenylate nucleotide charge remained unchanged in both age groups. Except for citrate, the metabolic reactions to arterial **hypoxemia** were qual. similar in both age groups, but they decreased with aging, obviously indicating reduced metabolic demands.

ST brain energy metab aging **hypoxemia**

IT Senescence and Senility
(carbohydrate and energy metab. by brain in, **hypoxia** in relation to)

IT Brain, metabolism
(carbohydrates and energy metab. by, aging and **hypoxia** effect on)

IT Carbohydrates and Sugars, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(metab. of, by brain, aging and **hypoxia** effect on)

IT 50-21-5, biological studies 50-99-7, biological studies 56-65-5, biological studies 56-73-5 57-04-5 58-64-0, biological studies 61-19-8, biological studies 67-07-2 77-92-9, biological studies 127-17-3, biological studies 328-50-7 488-69-7 643-13-0 6915-15-7
RL: BIOL (Biological study)
(of brain, aging and **hypoxia** effect on)

L47 ANSWER 14 OF 55 CAPLUS COPYRIGHT 2002 ACS

AN 1983:32487 CAPLUS

DN 98:32487

TI Influence of blood glucose concentration on brain lactate accumulation during severe **hypoxia** and subsequent recovery of brain energy metabolism

AU Gardiner, Mark; Smith, Maj Lis; Kaagstroem, Erik; Shohami, Esther; Siesjoe, Bo K.

CS Lab. Exp. Brain Res., Univ. Hosp., Lund, S-221 85, Swed.

SO J. Cereb. Blood Flow Metab. (1982), 2(4), 429-38
CODEN: JCBMDN; ISSN: 0271-678X

DT Journal

LA English

TI Influence of blood glucose concentration on brain lactate accumulation during severe **hypoxia** and subsequent recovery of brain energy metabolism

AB The effect of **hypoxemia** on regional cerebral blood flow (CBF)

and brain cortical metabolite concns. were investigated at different blood glucose concns. in rats under **N2O**-halothane anesthesia. Tissue **hypoxia** of 15-min duration was induced by a combination of arterial **hypoxemia**, hypotension, and clamping of the right carotid artery. Blood glucose concns. were manipulated by varying the food intake in the 24 h before the expt., and by glucose administration. Cortical CBF doubled during **hypoxia** on the intact side, but did not differ from control values on the clamped side. In the clamped hemisphere there was a substantial decrease in adenylate energy charge. At brain tissue glucose concns. of .gtoreq.1 .mu.mol/g, there was an inverse correlation between adenylate energy charge and brain lactate concn. In starved animals with mean brain glucose of 0.32 .mu.mol/g, lactate concn. was significantly lower, in spite of equally severe disruption of energy state. Recovery of brain adenylate energy charge was worse in fed and glucose-infused groups than in the fasted group. Thus, limitation of substrate supply during severe **hypoxia** in the rat allows enhanced recovery of brain energy metab. following the hypoxic episode.

ST brain **hypoxia** recovery glucose lactate

IT Blood sugar
(brain **hypoxia** recovery and energy metab. in relation to)

IT Glycolysis
(by brain, brain **hypoxia** recovery and energy metab. in relation to)

IT Brain, disease or disorder
(**hypoxia**, recovery from, blood sugar and brain lactate effect on brain energy metab. in relation to)

IT 73-24-5DP, nucleotides
RL: PRP (Properties); PREP (Preparation)
(energy charge of, of brain in **hypoxia** recovery, blood sugar and brain lactate in relation to)

IT 50-21-5P, biological studies
RL: BIOL (Biological study); PREP (Preparation)
(of brain, brain **hypoxia** recovery and energy metab. in relation to)

L47 ANSWER 15 OF 55 CAPLUS COPYRIGHT 2002 ACS

AN 1981:440025 CAPLUS

DN 95:40025

TI Interregional differences in brain intracellular pH and water compartmentation during acute normoxic and hypoxic hypocapnia in the anesthetized dog

AU Pelligrino, D. A.; Musch, T. I.; Dempsey, J. A.

CS Dep. Prev. Med., Univ. Wisconsin, Madison, WI, 53706, USA

SO Brain Res. (1981), 214(2), 387-404
CODEN: BRREAP; ISSN: 0006-8993

DT Journal

LA English

AB Interregional differences in intracellular pH (pHi) in brain tissue, and its regulation following 1 and 5 h of respiratory alkalosis (with and without **hypoxemia**) were detd. in **N2O** anesthetized dogs. Two techniques for pHi estn. were used (total CO2 (TCO2) and [14C]dimethadione) and included corrections for measured extracellular fluid (35SO42-) space (ECS). Cortical pHi by the 2 techniques agreed closely in control and in 3 of the 4 exptl. conditions, suggesting: (1) the estn. of extracellular fluid (ECF) HCO3- concn. from measured cerebrospinal fluid (CSF) HCO3- concn. was a valid assumption; and (2) the method had sufficient resoln. to det. the magnitude of brain pHi regulation during respiratory acid-base disturbances. When moderate

normoxic respiratory alkalosis (arterial pCO2 .apprx.25 torr) was imposed for 5 h, pHi (in most brain regions) was well regulated and always exceeded the incomplete regulation noted in bulk CSF. When moderate **hypoxemia** (arterial pO2 .apprx.45 torr) accompanied hypocapnia, pHi was more closely regulated during the early phase (1 h) of respiratory alkalosis. Increased levels of metabolic acids (esp. lactic acid) were crit. to brain pHi regulation during the initial hour of respiratory alkalosis, and accounted for much of the independent effect of **hypoxemia** on pHi regulation. However, these metabolic acids remained unchanged as pHi was more completely regulated between 1 and 5 h of continued hypocapnia or hypoxic hypocapnia. This time-dependent regulation of pHi may involve some regulatory role for changed transmembrane fluxes of H+ and(or) HCO3-. Significant interregional differences were obsd. in both pHi and ECS with tendencies toward more alk. pHi and lower ECS in brain stem and white matter. With respiratory alkalosis ECS fell and intracellular fluid increased in both cortex and caudate nucleus, possibly reflecting an osmotic effect of increased metabolic acid levels or redn. in cell membrane ion pumping.

ST brain pH water hypocapnia **hypoxia**; alkalosis brain pH water

IT Hypocapnia
(water metab. by brain in, **hypoxia** in relation to)

IT Brain, metabolism
(water metab. by, in hypocapnia, **hypoxia** in relation to)

IT Alkalosis
(respiratory, water metab. by brain in, **hypoxia** in relation to)

IT 7732-18-5, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(metab. of, by brain in hypocapnia, **hypoxia** in relation to)

IT 50-21-5, biological studies 56-65-5, biological studies 56-84-8, biological studies 56-86-0, biological studies 58-64-0, biological studies 77-92-9, biological studies 127-17-3, biological studies 328-42-7 328-50-7 6915-15-7
RL: BIOL (Biological study)
(of brain, in hypocapnia, **hypoxia** in relation to)

L47 ANSWER 16 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1980:526559 CAPLUS
DN 93:126559
TI Nitrous oxide availability
AU Murray, Michael J.; Murray, William J.
CS Dep. Anesthesiol., Duke Univ. Med. Cent., Durham, NC, 27710, USA
SO J. Clin. Pharmacol. (1980), 20(4, Pt. 1), 202-5
CODEN: JCPCBR; ISSN: 0091-2700
DT Journal
LA English
AB **N2O** is marketed as an inhalation anesthetic and as a food ingredient (e.g., whipping cream propellant). In the human, inhalation was assocd. with highs, peripheral nerve damage, mitotic poisoning of bone marrow, psychosis, and mental impairment. Exposure to **hypoxemic** mixts. has resulted in death. The whipping cream aerosol cans, when not shaken, will dispense .gtoreq.3 L of 87-90% **N2O**. Charger misuse may occur when they are substituted for identically designed CO2 chargers of a seltzer bottle; 4.3-5.0 L of 93-8% **N2O** is expelled at a controllable rate. The toxicity of these inexpensive **N2O** products, their high potential for misuse, and the absence of labeling (chargers) argue that their distribution be discontinued.

L47 ANSWER 17 OF 55 CAPLUS COPYRIGHT 2002 ACS

AN 1978:400627 CAPLUS
DN 89:627
TI Cardio-respiratory effects of nitrous oxide:oxygen:halothane anesthesia administered to dental outpatients in the upright position
AU Al-Khishali, T.; Padfield, A.; Perks, E. R.; Thornton, J. A.
CS Dep. Oral Surg., Coll. Dent., Baghdad, Iraq
SO Anaesthesia (1978), 33(2), 184-8
CODEN: ANASAB; ISSN: 0003-2409
DT Journal
LA English
AB The cardiorespiratory responses of dental patients receiving 30% O with **N2O** and halothane [151-67-7] while seated upright are reported. A high degree of sympathetic autonomic activity was noted with considerable lability of the blood pressure and pulse rate. **Hypoxemia** caused by respiratory obstruction, unrecognized by the anesthetist, occurred in approx. 20% of the patients at the time of insertion of the prop or pack and during removal of teeth.

L47 ANSWER 18 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1977:495488 CAPLUS
DN 87:95488
TI **Hypoxia**-induced vasoconstriction in isolated perfused lungs exposed to injectable or inhalation anesthetics
AU Bjertnaes, Lars J.
CS Fysiol. Inst., Oslo, Norway
SO Acta Anaesthesiol. Scand. (1977), 21(2), 133-47
CODEN: AANEAB
DT Journal
LA English
TI **Hypoxia**-induced vasoconstriction in isolated perfused lungs exposed to injectable or inhalation anesthetics
AB The effects of inhalation and injectable anesthetics on the vasoconstrictor response to acute alveolar **hypoxia** were compared in isolated blood-perfused rat lungs. The response was unaffected by **N2O** and injectable anesthetics, whereas, a reversible, dose-dependent damping effect was demonstrated for the volatile inhalation anesthetics, ether [60-29-7], halothane [151-67-7], and methoxyflurane [76-38-0]. The effect was demonstrated at blood concns. comparable to those used in clin. anesthesia, and it was not due to a general paralysis of smooth muscle. The findings may explain the occurrence of arterial **hypoxemia** during general inhalation anesthesia.
ST anesthetic **hypoxia** vasoconstriction lung; ether alveolar **hypoxia** vasoconstriction; halothane alveolar **hypoxia** vasoconstriction; methoxyflurane alveolar **hypoxia** vasoconstriction
IT Blood vessel
(constriction of, in **hypoxia**, anesthetics effect on)
IT Anesthetics
(injectable, vasoconstriction in **hypoxia** response to)
IT Anesthetics
(vasoconstriction in **hypoxia** response to)
IT 60-29-7, biological studies 76-38-0 151-67-7
RL: BIOL (Biological study)
(vasoconstriction in **hypoxia** response to)
IT 50-09-9 57-33-0 71-73-8 359-83-1 437-38-7 439-14-5 548-73-2 6740-88-1 10024-97-2, biological studies
RL: BIOL (Biological study)
(vasoconstriction response to, in **hypoxia**)

L47 ANSWER 19 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1972:21196 CAPLUS
DN 76:21196
TI Effect of nitrous oxide on the pulmonary circulation during venous air embolism
AU Munson, Edwin S.
CS Sch. Med., Univ. California, Davis, Calif., USA
SO Anesth. Analg. (Cleveland) (1971), 50(5), 785-93
CODEN: AACRAT
DT Journal
LA English
AB Nitrous oxide [10024-97-2] ventilation (80% **N2O**-20% O; 250-350 ml/breath) of pentobarbital [57-33-0]-anesthetized dogs after air emboli injection (1.5 ml/kg) caused a rapid increase (.sim.30%) in pulmonary arterial pressure compared to air ventilated controls. **Hypoxemia**, present following air embolization, did not increase after **N2O** was administered, despite increased wasted ventilation and carbon dioxide [124-38-9] partial pressure. Should venous air embolism occur during anesthesia, elimination of **N2O** may provide a rapid, effective initial step in treatment of this complication.

L47 ANSWER 20 OF 55 CAPLUS COPYRIGHT 2002 ACS
AN 1964:33675 CAPLUS
DN 60:33675
OREF 60:6030e-h
TI Pathological investigation on the cerebral carbohydrate metabolism in essential hypertension and cerebral arteriosclerosis
AU Otani, Haruhiko
CS Osaka Med. Coll., Takatsuki
SO Japan. Circulation J. (1963), 27(7), 513-15,534-46,547-61
DT Journal
LA Unavailable
AB The cerebral carbohydrate metabolism of patients with pulmonary tuberculosis, hypertension, or cerebral arteriosclerosis was measured during a resting state and during **hypoxemia** induced by 10% O inhalation for 20 min. against healthy controls. A catheter was introduced into the bulbus venae jugularis interna and the cerebral blood flow detd. by the **N2O** method. Glucose, lactate, and pyruvate levels of the arterial and internal jugular venous bloods were detd. In cerebral arteriosclerosis and pulmonary tuberculosis, the uptake and utilization of C3 acids of patients in a resting state in brain tissues were observed. It is concluded that the disturbance in the pathways of cerebral carbohydrate metabolism is in the anaerobic glycolysis rather than in the tricarboxylic acid cycle. In hypertension and esp. cerebral arteriosclerosis in induced **hypoxemia**, the disturbance of the cerebral carbohydrate metabolism is due to poor cerebral vascular response and disturbance of the tricarboxylic acid cycle, which occur in all groups. In some cases with cerebral arteriosclerosis during induced **hypoxemia** disturbance in consciousness was observed. Rapid decrease of cerebral O consumption to 1/2-1/3 of the resting state and release rather than utilization of glucose were observed. This is thought to be due to the increased disturbance in the anaerobic glycolysis which causes a marked decrease in the carbohydrate energy utilization. Significantly, those cases without any disturbance in consciousness showed less severe disturbance in O and carbohydrate utilization.

L47 ANSWER 21 OF 55 MEDLINE
AN 2001093787 MEDLINE
DN 21026490 PubMed ID: 11153632

TI Peritoneal ventilation with oxygen improves outcome after hemorrhagic shock in rats.
AU Barr J; Prueckner S; Safar P; Tisherman S A; Radovsky A; Stezoski J; Eshel G
CS Pediatric Intensive Care Unit, Assaf Harofeh Medical Center, Sackler Faculty of Medicine, Tel Aviv University, Zerifin, Israel.
SO CRITICAL CARE MEDICINE, (2000 Dec) 28 (12) 3896-901.
Journal code: 0355501. ISSN: 0090-3493.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200101
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010125
AB OBJECTIVE: In experimental pulmonary consolidation with **hypoxemia** in rabbits, peritoneal ventilation (PV) with 100% oxygen (PV-O2) improved PaO2. We hypothesized that PV-O2 could improve outcome after hemorrhagic shock (HS) with normal lungs, by mitigating dysoxia of the abdominal viscera. DESIGN: Randomized, controlled, laboratory animal study. SETTING: University animal research facility. SUBJECTIVE: Male Sprague-Dawley rats. INTERVENTIONS: Thirty rats under light anesthesia (**N2O**/oxygen plus halothane) and spontaneous breathing underwent blood withdrawal of 3 mL/100 g over 15 mins. After volume-controlled HS phase 1 of 60 mins, resuscitation phase 2 of 60 mins included infusion of shed blood and, if necessary, additional lactated Ringer's solution intravenously to control normotension from 60 to 120 mins. This was followed by observation phase 3 for 7 days. We randomized three groups of ten rats each: group I received PV-O2, starting at 15 mins of HS at a rate of 40 inflations/min, and a peritoneal "tidal volume" of 6 mL, until the end of phase 2. Group II received the same PV with room air (PV-Air). Control group III was treated without PV. MEASUREMENTS AND MAIN RESULTS: During the second half of HS phase 1, mean arterial pressures were higher in the PV-O2 group I compared with the PV-Air group II and control group III ($p < .05$). All 30 rats survived the 120 mins of phases 1 and 2. Survival to 7 days was achieved by ten of ten rats in PV-O2 group I; by nine of ten in PV-Air group II; and by five of ten in control group III ($p < .05$ vs. group I; NS vs. group II). Survival times of <7 days were 5 days in the one death of group II and ranged between 6 hrs and 4 days in the five deaths of group III. In 7-day survivors, neurologic deficit scores (0% to 10% = normal, 100% = death) were normal, ranging between zero and 8%. Necropsies of rats that died during phase 3 showed multiple areas of necrosis of the gut, some with perforations. Necropsies in the five survivors to 7 days of group III showed marked macroscopic and microscopic changes (scattered areas of necrosis of stomach and intestine, adhesions, and pale areas in the liver). These changes were absent or less severe in the nine survivors of group II. Viscera appeared normal in all ten rats of PV-O2 group I. CONCLUSIONS: Peritoneal ventilation with oxygen during and after severe hemorrhagic shock in rats seems to decrease morbidity and mortality by helping preserve viability of abdominal viscera.

L47 ANSWER 22 OF 55 MEDLINE
AN 2000418524 MEDLINE
DN 20413908 PubMed ID: 10958035
TI Cardiac arrest induced by accidental inhalation of anoxic gases, is the cause always a lack of oxygen?.
AU Jawan B; Lee J H
CS Department of Anesthesiology, Chang Gung Memorial Hospital, Kaohsiung,

Taoyuan, R.O.C.. jawanb@hotmail.com
SO CHANG-KENG I HSUEH TSA CHIH, (2000 Jun) 23 (6) 331-8.
Journal code: 9809559.
CY CHINA (REPUBLIC: 1949-)
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200009
ED Entered STN: 20000915
Last Updated on STN: 20000915
Entered Medline: 20000907
AB BACKGROUND: We experienced a case of accidental administration of 100% carbon dioxide (CO2) during anesthesia, which resulted in cardiac arrest. After successful cardio-pulmonary resuscitation the child recovered without brain damage. This outcome was quite different than that of the more commonly reported accidental administration of 100% nituous oxide (**N2O**), as the latter usually results in death from cerebral damage rather than cardiac arrest. We speculated that the cause of death and/or cardiac arrest may differ between these two anoxic gases. METHODS: Fourteen dogs were anesthetized and divided into two groups to receive either 100% CO2 or 100% **N2O**. Blood pressure (BP), heart rate (HR), cardiac output (CO), dp/dt, pulmonary artery pressure (PAP), central venous pressure (CVP) and blood gases (BG) were measured every 30 seconds until cardiac arrest (CA) occurred. RESULTS: The CO2 group showed a rapid decline in BP, HR, dp/dt, CO, pH, and PaO2 and a rise in PAP, CVP, and PaCO2, with CA occurring at 119 +/- 41 seconds. At the time of CA, the BG values were pH 6.6 +/- 0.09, PaCO2 375 +/- 69, and PaO2 62 +/- 15 mm Hg. The **N2O** group maintained BP, HR, dp/dt, pH, PaCO2, and experienced a rapid decline in PaO2 as in the CO2 group until 180 seconds, at which time the PaO2 was 12.3 +/- 3 mm Hg. CA occurred at 390 +/- 52 seconds. The values for pH, PaCO2 and PaO2 were 7.5 +/- 0.05, 25 +/- 15 and 4.8 +/- 1 mm Hg, respectively, at the time of CA. CONCLUSION: One hundred percent CO2-induced cardiac arrest occurred in 119 seconds and was not oxygen-dependent, whereas 100% **N2O** induced cardiac arrest occurred in 390 seconds and was clearly dependent on **hypoxemia**.

L47 ANSWER 23 OF 55 MEDLINE
AN 2000031202 MEDLINE
DN 20031202 PubMed ID: 10566926
TI Unilateral negative pressure pulmonary edema during anesthesia with a laryngeal mask airway.
AU Sullivan M
CS Department of Anaesthesia and Intensive Care, Cairns Base Hospital, Queensland, Australia. kellmat.interlog.com.
SO CANADIAN JOURNAL OF ANAESTHESIA, (1999 Nov) 46 (11) 1053-6.
Journal code: 8701709. ISSN: 0832-610X.
CY Canada
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991201
AB PURPOSE: To present a case of unilateral pulmonary edema after upper airway obstruction. CLINICAL FEATURES: In a 21-yr-old man, anesthesia was induced with propofol and maintained with **N2O**/O2/isoflurane via an LMA. After being placed in the lateral position, he had an episode of upper airway obstruction while breathing spontaneously. **Hypoxemia**

(SpO2 80-83%) refractory to the administration of oxygen (F1O2 1.0) ensued following relief of the obstruction. Chest X-ray showed edema of the dependent lung. Treatment consisted of placing the patient in the sitting position and supplemental oxygen. The situation resolved over a few hours. CONCLUSION: If airway obstruction occurs in the lateral position, development of negative pressure pulmonary edema (NPPE) in the dependent lung is favoured by hydrostatic forces and possibly the elevated resting position of the dependent hemidiaphragm.

L47 ANSWER 24 OF 55 MEDLINE
AN 1999069799 MEDLINE
DN 99069799 PubMed ID: 9852697
TI Pulmonary edema due to acute airway obstruction immediately after tracheal extubation.
AU Kadota Y; Imabayashi T; Gushiken T; Kawasaki K; Oda T; Yoshimura N
CS Department of Anesthesiology & Critical Care Medicine, Kagoshima University School of Medicine.
SO MASUI. JAPANESE JOURNAL OF ANESTHESIOLOGY, (1998 Nov) 47 (11) 1333-7. Journal code: 0413707. ISSN: 0021-4892.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA Japanese
FS Priority Journals
EM 199903
ED Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990317
AB A 33-year-old male was scheduled for tonsillectomy and pharyngoplasty due to sleep apnea syndrome. The intubation was uneventful following induction with thiamylal and vecuronium. Anesthesia was maintained with O2-**N2O**-sevoflurane. No complications were observed during the 90 min operation. After the termination of the anesthesia, a hyperadrenergic state was observed: arterial pressure and heart rate rose to 230/135 mmHg and 135 bpm, respectively. Immediately after extubation, he developed dyspnea with tracheal tag and stridor, and became cyanotic despite the use of a simple oxygen mask and assisted ventilation. Laryngospasm was suspected. The patient was reintubated and suctioned; pink, frothy sputum was not obtained. Arterial blood gases 5 minutes after reintubation revealed a pH of 7.24, Pao2 86 mmHg (FIo2 1.0), and Paco2 54 mmHg. Chest X-ray 30 minutes after reintubation revealed bilateral diffuse alveolar infiltration. The diagnosis was interstitial pulmonary edema. The patient was ventilated mechanically by applying a positive end-expiratory pressure of 5cm H2O, and furosemide and dopamine were administered intravenously. The patient was extubated the next day, and discharged from hospital ten days later. We considered that the lung edema was induced by the severe negative pressure generated by inspirating against a closed upper airway, as well as by the hyperadrenergic state and severe **hypoxemia** observed during and after extubation.

L47 ANSWER 25 OF 55 MEDLINE
AN 1999020271 MEDLINE
DN 99020271 PubMed ID: 9803430
TI Upper airway obstruction during midazolam/nitrous oxide sedation in children with enlarged tonsils.
AU Litman R S; Kottra J A; Berkowitz R J; Ward D S
CS Division of Pediatric Anesthesia, University of Rochester, New York, USA.
SO PEDIATRIC DENTISTRY, (1998 Sep-Oct) 20 (5) 318-20. Journal code: 7909102. ISSN: 0164-1263.
CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Dental Journals; Priority Journals
EM 199901
ED Entered STN: 19990202
Last Updated on STN: 19990202
Entered Medline: 19990120
AB PURPOSE: The purpose of this nonrandomized, case-control study was to examine the incidence and severity of upper airway obstruction (UAO) in children with enlarged tonsils during inhalation of nitrous oxide (**N2O**). METHODS: Following premedication with oral midazolam, 0.5 mg/kg, measurements were collected during a 3-minute control period followed by 3 minutes of breathing 50% **N2O** in oxygen. An unblinded anesthesiologist held a facemask over the child's mouth and nose without supporting the head or neck, or attempting to maintain airway patency. Every 20 seconds, the degree of airway obstruction was graded as none, partial, or complete. Twenty-five children presenting for tonsillectomy and 25 controls without enlarged tonsils participated. RESULTS: During 50% **N2O** inhalation, 14 children (56%) in the tonsillectomy group, and four children (16%) in the control group demonstrated partial UAO. One child in the tonsillectomy group with partial UAO developed **hypoxemia** (SpO2 72%). One child in the tonsil group developed complete UAO during inhalation of 50% **N2O** . CONCLUSION: Children who receive sedation with oral midazolam and 50% **N2O** inhalation may exhibit significant UAO, especially in the presence of enlarged tonsils. Presedation physical exams should evaluate the presence of tonsil size during examination of the mouth and airway.

L47 ANSWER 26 OF 55 MEDLINE
AN 1998055113 MEDLINE
DN 98055113 PubMed ID: 9393395
TI Breathing patterns and levels of consciousness in children during administration of nitrous oxide after oral midazolam premedication.
AU Litman R S; Kottra J A; Berkowitz R J; Ward D S
CS University of Rochester School of Medicine and Dentistry, NY, USA.. RLitman@anes.rochester.edu
SO JOURNAL OF ORAL AND MAXILLOFACIAL SURGERY, (1997 Dec) 55 (12) 1372-7; discussion 1378-9.
Journal code: 8206428. ISSN: 0278-2391.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Dental Journals; Priority Journals
EM 199712
ED Entered STN: 19980116
Last Updated on STN: 19990129
Entered Medline: 19971229
AB PURPOSE: The combination of midazolam and nitrous oxide is commonly used to achieve sedation and analgesia during pediatric oral procedures, yet there are few, if any, data that illustrate the ventilatory effects of **N2O** in children, especially when used in combination with additional central nervous system (CNS) depressants. It was hypothesized that the addition of **N2O** inhalation to oral midazolam premedication would enhance the sedative effects of the midazolam and add analgesia without causing significant respiratory depression. The purpose of this study was to test this hypothesis. MATERIALS AND METHODS: Thirty-four healthy children about to undergo restorative dental treatment under general anesthesia were premedicated with oral midazolam, 0.7 mg/kg, and were then exposed to 40% **N2O** for 15 minutes after a 5-minute

control period. The effect of adding **N2O** on SpO2, respiratory rate, PETCO2, VT, and VT/TI was examined and the levels of consciousness (conscious vs deep sedation) before and during **N2O** inhalation were determined. RESULTS: During the course of the study, no child developed **hypoxemia** (SpO2 < 92%) nor clinically significant upper airway obstruction. Four children who did not develop hypoventilation (defined as PETCO2 > 45 mm Hg) during the control period did so after initiation of **N2O**. Overall, there were no significant differences in SpO2, PETCO2, VT, or VT/TI between the control and study periods. However, respiratory rates were significantly higher in the first 10 minutes of **N2O** inhalation when compared with the control period. Before starting **N2O** administration, 14 children were not clinically sedated, 19 children met the criteria for conscious sedation, and one child met the criteria for deep sedation. At the end of 15 minutes of **N2O** inhalation, 12 children were not clinically sedated, 17 children met the definition of conscious sedation, three were deeply sedated, and one child had no response to IV insertion, implying a state of general anesthesia. There were no differences in sedation scores between the control and study periods (P = .6). Overall, seven children had an increase in their sedation score while breathing **N2O**, four had a decrease in their sedation score, and 22 had no change. CONCLUSIONS: The addition of 40% **N2O** to oral midazolam, 0.7 mg/kg, did not result in clinically meaningful respiratory depression nor upper airway obstruction, but did, in some children, cause an increase in the level of sedation beyond simple conscious sedation.

L47 ANSWER 27 OF 55 MEDLINE
AN 96290450 MEDLINE
DN 96290450 PubMed ID: 8673188
TI Levels of consciousness and ventilatory parameters in young children during sedation with oral midazolam and nitrous oxide.
CM Comment in: Arch Pediatr Adolesc Med. 1996 Jul;150(7):665-7
AU Litman R S; Berkowitz R J; Ward D S
CS Department of Pediatrics, University of Rochester School of Medicine and Dentistry, NY, USA.. Rlitman@ccmail.anes.rochester.edu
SO ARCHIVES OF PEDIATRICS AND ADOLESCENT MEDICINE, (1996 Jul) 150 (7) 671-5. Journal code: 9422751. ISSN: 1072-4710.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199608
ED Entered STN: 19960822
Last Updated on STN: 19980206
Entered Medline: 19960815
AB OBJECTIVE: To determine the ventilatory effects and levels of consciousness achieved during sedation with the combination of oral midazolam and inhaled nitrous oxide. DESIGN: Case series. SETTING: Surgical suite. PATIENTS: Twenty-two consecutive children, aged 1 to 3 years, were seen for elective, ambulatory surgery. INTERVENTIONS: Patients were premedicated with oral midazolam hydrochloride, 0.5 mg/kg, and then breathed 4 concentrations of nitrous oxide (**N2O**) in oxygen (15%, 30%, 45%, and 60%) for 4 minutes at each concentration prior to induction of general anesthesia. MAIN OUTCOME MEASURES: Levels of consciousness (conscious vs deep sedation) and ventilatory parameters: respiratory rate, end-tidal carbon dioxide tension (PETCO2), and oxyhemoglobin saturation (SPO2). Upper airway obstruction was diagnosed by clinical assessment by an experienced pediatric anesthesiologist (R.S.L.) and respiratory impedance plethysmography. RESULTS: During inhalation of **N2O**, 12

of the 20 children demonstrated a mild degree of ventilatory depression; PETCO2 values were equal to or greater than 45 mm Hg during at least 2 concentrations of **N2O** studied. There were no significant changes in SPO2 or PETCO2 with increasing concentrations of **N2O** (P > .05). Respiratory rates tended to be lower during inhalation of 15% **N2O** than at higher concentrations (P = .05). No child developed upper airway obstruction or **hypoxemia** (SPO2 < 92%) at any level of **N2O** inhalation. Sedation scores were significantly higher at 60% **N2O** than at all other concentrations of **N2O** (P < .02) At 15% **N2O**, 12 children were not clinically sedated, 8 children met the American Academy of Pediatrics definition of conscious sedation, and no child met the definition of deep sedation. At 30% **N2O**, 10 children were not clinically sedated, 9 met the definition of conscious sedation, and 1 child met the definition of deep sedation. At 45% **N2O**, 9 children were not clinically sedated, 9 met the definition of conscious sedation, and 2 met the definition of deep sedation. At 60% **N2O**, 6 children were not clinically sedated, 6 met the definition of conscious sedation, 6 met the definition of deep sedation, and 1 child progressed to a deeper level of sedation in that there was no response to a painful stimulus. One child was withdrawn from the study during inhalation of 45% **N2O** because of emesis. CONCLUSIONS: The combination of oral midazolam, 0.5 mg/kg, and up to 60% inhaled **N2O** caused mild ventilatory depression in some children and resulted in a progression from conscious to deep sedation beginning at 30% **N2O**. When using this particular combination of sedatives, practitioners should monitor each child's mental status continuously and adhere to the appropriate published guidelines for the monitoring and management of such patients.

L47 ANSWER 28 OF 55 MEDLINE
AN 95290211 MEDLINE
DN 95290211 PubMed ID: 7772362
TI A random trial comparing recovery after midazolam-alfentanil anesthesia with and without reversal with flumazenil, and standardized neurolept anesthesia for major gynecologic surgery.
AU Jensen A G; Moller J T; Lybecker H; Hansen P A
CS Department of Anaesthesia, Esbjerg Central Hospital, Denmark.
SO JOURNAL OF CLINICAL ANESTHESIA, (1995 Feb) 7 (1) 63-70.
Journal code: 8812166. ISSN: 0952-8180.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 199507
ED Entered STN: 19950720
Last Updated on STN: 19950720
Entered Medline: 19950713
AB STUDY OBJECTIVE: To compare the recovery characteristics of total intravenous anesthesia (TIVA) using midazolam-alfentanil, with or without reversal with flumazenil to a standardized neurolept anesthesia with nitrous oxide (**N2O**). DESIGN: Randomized, double-blinded clinical study. SETTING: University medical center. PATIENTS: 80 ASA physical status I and II women scheduled for major elective gynecologic surgery. INTERVENTIONS: Patients were anesthetized with one of three different anesthetic techniques. Patients in the TIVA group with reversal received midazolam-alfentanil reversed with flumazenil (Group 1), the TIVA group without reversal received midazolam-alfentanil reversed with placebo

(Group 2), and patients in the neurolept group received anesthesia using thiopental sodium, droperidol, fentanyl, and **N2O** (Group 3). MEASUREMENTS AND MAIN RESULTS: Recovery was assessed by an observer blinded to the treatment allocation, using a Modified Steward Recovery Score and judgment of orientation and comprehension, collaboration and degree of sedation for the first 4 hours after extubation. Arterial blood gases were measured 30 minutes after extubation. A questionnaire regarding the degree of perioperative amnesia was presented to the patients 4 and 24 hours after surgery. The recovery scores were better in the TIVA group with reversal than in the other two groups from 0 to 30 minutes postoperatively. No difference between the groups could be found thereafter, although after 30 minutes some resedation occurred in the TIVA group with reversal. The median injected amount of flumazenil in Group 1 was 0.5 mg. Respiratory depression (breathing frequency below 10 breaths/min) was reversed with naloxone in one patient in the TIVA group with reversal, five patients in the TIVA group without reversal, and no patient in the neurolept group ($p < 0.001$). On blood gas analysis, there was no evidence of **hypoxemia** or carbon dioxide retention. No difference was seen between the groups regarding consumption of analgesics, degree of amnesia, or patient rating of the quality of anesthesia. One patient in Group 2, however, recorded awareness at skin incision when questioned 4 hours after the operation, but could not recall this 20 hours later. CONCLUSIONS: TIVA with midazolam and alfentanil can be used for major gynecologic surgery. Recovery in the neurolept group was equal to recovery in the TIVA group without reversal, and flumazenil improves the recovery after midazolam anesthesia. Overall, in comparison with the neurolept technique no major advantage could be demonstrated using TIVA with midazolam-alfentanil.

L47 ANSWER 29 OF 55 MEDLINE
AN 94325085 MEDLINE
DN 94325085 PubMed ID: 8049053
TI Laryngeal mask airway vs face mask and Guedel airway during pediatric myringotomy.
AU Watcha M F; Garner F T; White P F; Lusk R
CS Department of Anesthesiology and Pain Management, University of Texas Southwestern Medical Center at Dallas.
SO ARCHIVES OF OTOLARYNGOLOGY -- HEAD AND NECK SURGERY, (1994 Aug) 120 (8) 877-80.
Journal code: 8603209. ISSN: 0886-4470.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199409
ED Entered STN: 19940914
Last Updated on STN: 19980206
Entered Medline: 19940906
AB OBJECTIVE: To compare perioperative conditions when a face mask and Guedel oral airway (FM-OA) or a laryngeal mask airway (LMA) are used to maintain airway patency during bilateral myringotomy with insertion of tympanostomy tubes (BMT). DESIGN: Randomized controlled trial in children's hospital tertiary-care operating rooms. PARTICIPANTS: Fifty healthy children undergoing BMT procedures during halothane--nitrous oxide (**N2O**) anesthesia. INTERVENTIONS: During BMT we managed the airway by inserting a Guedel oral airway or an LMA. MAIN OUTCOME MEASURES: We recorded the time taken to insert the airway device along with oxygen saturation during the

operation and time from the end of surgery to eye opening, response to commands, and home readiness. In addition the surgeon assessed perioperative conditions on a 10-point scale (1, poor, through 10, excellent). RESULTS: Although insertion of the LMA took longer than the Guedel oral airway (mean +/- SD, 9 +/- 2 seconds vs 6 +/- 2 seconds; P < .05), no differences were noted in the actual operating, anesthesia, or recovery times. However, the frequency of **hypoxemic** episodes was decreased (8% vs 36%, P < .05) and the lowest recorded oxygen saturations were higher (mean +/- SD, 95% +/- 7% vs 88% +/- 12%; P < .05) in the LMA group than in the FM-OA group. Surgeons rated perioperative conditions better when the LMA was used (median score, 9 vs 8; P < .05). CONCLUSION: The LMA is an excellent alternative to the FM-OA technique for airway maintenance in children undergoing BMT procedures during halothane--**N2O** anesthesia.

L47 ANSWER 30 OF 55 MEDLINE
AN 94069023 MEDLINE
DN 94069023 PubMed ID: 8248606
TI [Anesthesia recovery, gas exchange and postoperative hepatic and renal function in patients with morbid obesity undergoing bariatric surgery: comparison of the effects of halothane, isoflurane and fentanyl]. Recuperacion anestesica, intercambio gaseoso y funcion hepatica y renal postoperatorios en pacientes con obesidad morbida sometidos a cirugia bariatrica: comparacion de los efectos del halotano, isoflurano y fentanilo.
AU Melero A; Valles J; Vila P; Canet J; Vidal F
CS Servicio de Anestesiologia y Reanimacion, Hospital Germans Trias i Pujol, Badalona.
SO REVISTA ESPANOLA DE ANESTESIOLOGIA Y REANIMACION, (1993 Sep-Oct) 40 (5) 268-72.
Journal code: 0134516. ISSN: 0034-9356.
CY Spain
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA Spanish
FS Priority Journals
EM 199312
ED Entered STN: 19940201
Last Updated on STN: 19950206
Entered Medline: 19931223
AB OBJECTIVES. To compare the postoperative effects of three anesthetic agents, fentanyl, halothane and isoflurane, on recovery from anesthesia, changes in arterial blood gases, and tests of liver and kidney function in morbidly obese patients recovering from vertical ring gastroplasty. MATERIAL AND METHODS. Thirty-three patients were studied, randomly distributed into three groups of 11. Induction for all was with atracurium (5 mg), 2.5% thiopentone sodium (5-6 mg.kg-1), succinylcholine (1.5 mg.kg-1) and orotracheal intubation. Anesthesia was maintained with intermittent doses of fentanyl (group F), 2% halothane (group H) or 2.5% isoflurane (group I). All patients received a 50% O2/**N2O** mixture at a minute volume calculated on ideal weight. Muscle relaxation was achieved by continuous perfusion of atracurium. Postoperative analgesia was by morphine chloride through a lumbar epidural catheter. Time of eye opening and time of extubation were recorded. Arterial blood gas measurements were taken and the results of liver and kidney function tests were recorded until the 7th day after surgery. RESULTS. Eye opening after awakening was earlier in the fentanyl group (6 +/- 5 min), but no differences were found for time of extubation. Blood gas measurements for

the 33 patients revealed a significant decrease in PaO2 (58 +/- 14 mmHg), a slight increase of PaCO2 (40 +/- 6 mmHg) and a lower pH (7.32 +/- 0.04) immediately after surgery. On day seven, PaO2 had not yet reached preoperative levels ($p < 0.01$). These results were independent of anesthetic agent used. Kidney function tests showed significant rises in SGOT (81 +/- 36 U/l), SGPT (150 +/- 110 U/l) and bilirubin (Bil: 15 +/- 5 mmol/l) and decreases in prothrombin activity (PT: 73 +/- 11%) 24 hours after surgery, with later normalization. Urea fell significantly throughout the seven-day period (3.2 +/- 1.3 mmol/l). These results were also independent of the anesthetic agent used. CONCLUSIONS. Morbidly obese patients undergoing gastroplasty recover from anesthesia in the same way regardless of the agent used. The early postoperative period is characterized by severe **hypoxemia** and transitory changes in kidney function tests. Neither of these findings is dependent on the agent used.

L47 ANSWER 31 OF 55 MEDLINE
AN 93249059 MEDLINE
DN 93249059 PubMed ID: 8484513
TI Ventilation with nitrous oxide during open cholecystectomy increases the incidence of postoperative **hypoxemia**.
AU Maroof M; Khan R M; Siddique M
CS Department of Anesthesiology, King Fahad National Guard Hospital, Riyadh, Kingdom of Saudi Arabia.
SO ANESTHESIA AND ANALGESIA, (1993 May) 76 (5) 1091-4.
Journal code: 1310650. ISSN: 0003-2999.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199306
ED Entered STN: 19930618
Last Updated on STN: 19930618
Entered Medline: 19930603
TI Ventilation with nitrous oxide during open cholecystectomy increases the incidence of postoperative **hypoxemia**.
AB The effect of intraoperative use of air versus nitrous oxide (**N2O**) on postoperative oxygen (O2) saturation in blood was evaluated in 40 ASA Class I and II patients undergoing elective, open cholecystectomy. Patients were allocated randomly to two groups on the basis of whether they received air (Group A, n = 20) or **N2O** (Group B, n = 20) intraoperatively. Oxygen saturation was recorded on arrival of the patients in the ward, 24 h, and 48 h postoperatively. Although mean O2 saturation did not differ significantly ($P > 0.05$) between the groups over the first 24 h postoperatively, it was significantly higher ($P < 0.05$) in Group A as compared to Group B 48 h postoperatively. Incidence of **hypoxemia** (O2 saturation < 90%) was 40% in Group B as compared to 0% in Group A at the end of 48 h postoperatively. We conclude that the use of **N2O** during cholecystectomy is associated with a higher incidence of **hypoxemia** postoperatively.

L47 ANSWER 32 OF 55 MEDLINE
AN 93225223 MEDLINE
DN 93225223 PubMed ID: 8468787
TI Perioperative pulmonary thromboembolism. A clinical study.
AU Ishizawa Y; Dohi S
CS Department of Anesthesiology, University of Tsukuba, School of Medicine.

SO MASUI. JAPANESE JOURNAL OF ANESTHESIOLOGY, (1993 Mar) 42 (3) 417-22.
Journal code: 0413707. ISSN: 0021-4892.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA Japanese
FS Priority Journals
EM 199305
ED Entered STN: 19930521
Last Updated on STN: 19930521
Entered Medline: 19930513
AB Pulmonary embolism (PE) is a major catastrophe during postoperative period. We had six patients who developed PE after surgery and one during anesthesia and surgery. Severe arterial **hypoxemia** (PaO2 41 +/- 14 mmHg) occurred in all six postoperative patients, but not in a patient who developed PE under anesthesia. In 3 patients with pulmonary artery catheter in place, pulmonary arterial pressure (PAP) increased significantly during the embolic events. PAP tended to decrease before the apparent improvement of PaO2 in each patient. This suggests that increases in anastomotic bronchial blood flow occurred following the events. In a patient who developed PE under enflurane-**N2O**-O2 anesthesia, neither **hypoxemia** nor hypotension occurred despite significant increase in PAP. All patients received heparin and urokinase intravenously, which caused persistent bleeding in two patients. It remains for further investigations to study the mechanisms of serious **hypoxemia** in postoperative patients with PE as well as those of favorably maintained pulmonary oxygenation in a patient with PE under general anesthesia.

L47 ANSWER 33 OF 55 MEDLINE
AN 92172429 MEDLINE
DN 92172429 PubMed ID: 1540364
TI Large visible gas bubbles in the internal jugular vein: a common occurrence during supine radical neck surgery?.
AU Rice J H; Gonzalez R M
CS Department of Anesthesiology, Eye and Ear Hospital, University of Pittsburgh, PA 15213.
SO JOURNAL OF CLINICAL ANESTHESIA, (1992 Jan-Feb) 4 (1) 21-4.
Journal code: 8812166. ISSN: 0952-8180.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199204
ED Entered STN: 19920424
Last Updated on STN: 19920424
Entered Medline: 19920409
AB STUDY OBJECTIVE: To establish the frequency of large visible bubbles or collections of bubbles in the jugular vein during radical neck dissection in the supine position. DESIGN: Prospective observation by at least two investigators of random consecutive patients scheduled for radical neck surgery. SETTING: Operating room suite in a university hospital specializing in head and neck cancer surgery. PATIENTS: Twelve consecutive ASA physical status II and III patients undergoing modified radical dissection for cancer. INTERVENTIONS: General anesthesia with fentanyl, oxygen (O2) 30% to 40%, nitrous oxide (**N2O**) 60% to 70%, and isoflurane 0.5% to 1.5%, with mechanical ventilation. Table position horizontal. Modified radical neck dissections performed by attending surgeons. Surgical field (jugular vein) carefully observed by the surgeons and an independent anesthesiologist investigator for the presence of

bubbles during the dissection. MEASUREMENTS AND MAIN RESULTS: Easily visible bubbles were observed in the jugular veins of 42% (5 of 12) of the consecutive radical neck dissection patients studied. Some of the collections of bubbles were large (greater than 2.5 cm in diameter). In one case, the appearance and subsequent disappearance of bubbles was followed by a transient drop in arterial blood pressure (BP) and in end-tidal carbon dioxide (PETCO2), which was suggestive of a diagnosis of central venous air embolization. CONCLUSIONS: We theorize that some unexplained, undesirable intraoperative events (hypotension, arrhythmia, and **hypoxemia**) during radical neck dissection could be a result of venous air embolus or paradoxical air embolus. The anesthesia community should be aware of the high frequency of these visible bubbles in the jugular veins during radical neck surgery, even in the supine position. At minimum, this phenomenon is a frequent event of intellectual interest. At worst, these bubbles may be harbingers of significant central air embolism.

L47 ANSWER 34 OF 55 MEDLINE
AN 91052462 MEDLINE
DN 91052462 PubMed ID: 2240630
TI Postoperative **hypoxemia** after nonabdominal surgery: a frequent event not caused by nitrous oxide.
AU Lampe G H; Wauk L Z; Whitendale P; Way W L; Kozmary S V; Donegan J H; Eger E I 2nd
CS Department of Anesthesia, University of California, San Francisco 94143-0464.
NC PO1 AG03104A (NIA)
SO ANESTHESIA AND ANALGESIA, (1990 Dec) 71 (6) 597-601.
Journal code: 1310650. ISSN: 0003-2999.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199012
ED Entered STN: 19910208
Last Updated on STN: 19950206
Entered Medline: 19901221
TI Postoperative **hypoxemia** after nonabdominal surgery: a frequent event not caused by nitrous oxide.
AB We tested whether anesthesia that includes nitrous oxide (**N2O**) results in the development of intraoperative and postoperative pulmonary complications, including **hypoxemia**. We also tested whether aging contributes to the development of such complications, particularly when anesthesia includes **N2O**. We randomly allocated patients having total hip replacements, carotid endarterectomies, or transsphenoidal hypophysectomies (total n = 270) to an anesthetic regimen with and without **N2O** (stratified within surgical group). A heat-and-moisture exchanger was included in the anesthetic circuit of all patients. Patients were monitored perioperatively and for 1 wk after surgery using intermittent and continuous pulse oximetry to determine oxyhemoglobin saturation. Intraoperatively, mean oxygen (O2) saturations were lower in patients given **N2O**, particularly older patients. **Hypoxemia** (O2 saturation less than 86%) developed in five patients receiving **N2O** and in one receiving O2. This difference was not significant. Administration of **N2O** did not decrease postoperative O2 saturation, nor did it alter the incidence of postoperative **hypoxemia**, cough, or sputum production.

L47 ANSWER 35 OF 55 MEDLINE
AN 91052460 MEDLINE
DN 91052460 PubMed ID: 2240628
TI Effect on outcome of prolonged exposure of patients to nitrous oxide.
AU Lampe G H; Wauk L Z; Donegan J H; Pitts L H; Jackler R K; Litt L L; Rampil I J; Eger E I 2nd
CS Department of Anesthesia, University of California, San Francisco 94143-0464.
NC PO1 AG03104A (NIA)
SO ANESTHESIA AND ANALGESIA, (1990 Dec) 71 (6) 586-90.
Journal code: 1310650. ISSN: 0003-2999.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199012
ED Entered STN: 19910208
Last Updated on STN: 19950206
Entered Medline: 19901221
AB Prolonged (several days or repeated) exposure to nitrous oxide (**N2O**) can cause injury or death. To assess whether relatively prolonged anesthesia with **N2O** in normal patients might similarly cause untoward effects, we investigated whether the addition of **N2O** to isoflurane anesthesia caused injury to patients having surgical resection of acoustic neuroma lasting approximately 10 h. Twenty-six patients undergoing surgical resection of acoustic neuroma were randomly assigned to a regimen that included or excluded **N2O** (50%-60%) during isoflurane anesthesia plus intravenous adjuvants. On average, slightly less isoflurane (0.24%) was used during anesthesia with **N2O**. We measured standard clinical variables (blood pressure, heart rate), oxygen saturation, neurologic status, pain, and the incidence and type of morbid outcomes. Exposure to **N2O** did not increase the incidence of morbid outcomes (including hepatic injury, infection, or **hypoxemia**), prolong hospitalization, or increase common postoperative complaints such as nausea, vomiting, coughing, or headache. Patients anesthetized with either regimen were equally satisfied with their anesthetic.

L47 ANSWER 36 OF 55 MEDLINE
AN 91052459 MEDLINE
DN 91052459 PubMed ID: 2240627
TI Clinical pharmacology of nitrous oxide: an argument for its continued use.
AU Eger E I 2nd; Lampe G H; Wauk L Z; Whitendale P; Cahalan M K; Donegan J H
CS Department of Anesthesia, University of California, San Francisco 94143-0464.
NC PO1 AG03104A (NIA)
SO ANESTHESIA AND ANALGESIA, (1990 Dec) 71 (6) 575-85.
Journal code: 1310650. ISSN: 0003-2999.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199012
ED Entered STN: 19910208

Last Updated on STN: 19950206
Entered Medline: 19901221

AB We tested the hypothesis that the administration of nitrous oxide (**N2O**) causes major (e.g., myocardial infarction, neuronal injury, **hypoxemia**, infection, death) or minor (e.g., nausea, vomiting, headache, earache) untoward effects in patients requiring anesthesia for 1.5-4 h. Given the higher morbidity and mortality associated with aging, we also tested whether aging increased any untoward effect of **N2O** . Finally, we investigated whether the substitution of **N2O** for a fraction of the anesthesia supplied by isoflurane altered the latter's pharmacologic effects. We studied 270 patients scheduled for elective total hip arthroplasty (n = 100), carotid endarterectomy (n = 70), or transsphenoidal hypophysectomy (n = 100) who were randomly assigned within each surgical group to receive isoflurane with or without 60% **N2O** . Regardless of patient age, we found no difference in major or minor untoward outcomes between anesthetic groups, nor a trend to suggest that a larger data cohort would reveal a significant adverse effect of **N2O**. The addition of **N2O** administration decreased the isoflurane requirement for clinical anesthesia but did not alter most of the clinical variables measured in practice, including blood pressure, heart rate, rate of recovery from anesthesia, development of postoperative pain, patient satisfaction with anesthesia, or duration of anesthesia or of hospitalization. Patients given **N2O** were no more likely to dream during anesthesia, remember events during anesthesia, or be frightened by those events. Our results support the continued use of **N2O** to anesthetize patients for elective surgery.

L47 ANSWER 37 OF 55 MEDLINE
AN 91039885 MEDLINE
DN 91039885 PubMed ID: 2232130
TI Anesthetic experience of a patient for splenectomy with severe liver dysfunction and hyperammonemia.
AU Kobayashi S; Sato Y; Kawate M; Yoshikawa H
CS Department of Anesthesiology, Toranomon Hospital, Tokyo.
SO MASUI. JAPANESE JOURNAL OF ANESTHESIOLOGY, (1990 Aug) 39 (8) 1033-9. Journal code: 0413707. ISSN: 0021-4892.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA Japanese
FS Priority Journals
EM 199012
ED Entered STN: 19910208
Last Updated on STN: 19910208
Entered Medline: 19901207

AB A case of a patient with severe liver dysfunction and hyperammonemia undergoing splenectomy and liver biopsy was reported. Preoperative examination revealed that this patient's liver function was severely impaired due to liver cirrhosis (ICG15 = 60%, HPT = 29%, serum NH3 = 110 micrograms.dl-1). Preoperatively, kanamycin 2 g.day-1 and lacturose 60 ml.day-1 were given and FFP 3-5 units.day-1 were infused. With no premedication, general anesthesia was induced with dTc 3 mg, thiopental 200 mg and SCC 80 mg. Anesthesia was maintained with **N2O** -O2-enflurane and pancuronium. Though **N2O** concentration was kept at 50% to prevent intraoperative **hypoxemia**, the necessary enflurane concentration was low (almost 1% or lower). Serum NH3 level during operation was stable (100-110 micrograms.dl-1), and the level decreased (66-90 micrograms.dl-1) postoperatively. Postoperatively, this patient's consciousness level fluctuated with or without flapping tremor. The treatment of hepatic encephalopathy with lactulose, aminoleban EN and

maalox were effective. Problems of perioperative and anesthetic management of a patient for upper abdominal surgery with severe liver dysfunction associated with hyperammonemia were discussed.

L47 ANSWER 38 OF 55 MEDLINE
AN 91012945 MEDLINE
DN 91012945 PubMed ID: 2214125
TI Changes in arterial oxygen tension during and after enflurane or halothane anesthesia as well as epidural analgesia.
AU Hoshi K; Shima T; Andoh K; Matsukawa S; Hashimoto Y
CS Department of Anesthesiology, Tohoku University School of Medicine, Sendai.
SO MASUI. JAPANESE JOURNAL OF ANESTHESIOLOGY, (1990 Jul) 39 (7) 910-4. Journal code: 0413707. ISSN: 0021-4892.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA Japanese
FS Priority Journals
EM 199011
ED Entered STN: 19910117
Last Updated on STN: 19910117
Entered Medline: 19901102
AB Recovery from inhalation anesthesia is often marked by the occurrence of postoperative **hypoxemia**. In this study, we compared the effects of enflurane or halothane anesthesia and epidural analgesia on arterial oxygen tension during and after the operation in 60 ASA physical status 1-2 patients who underwent cholecystectomy. Anesthesia was induced with thiopental and maintained with 66% **N2O** and -enflurane (1.5%), -halothane (1%), or -epidural lidocaine (1% solution, 17.5 ml) in oxygen. Blood gas analysis was done before and 10, 30, 60 min after induction. PaO2 was measured on 1st and 3rd postoperative days in all patients breathing air spontaneously. PaO2 decreased during operation in all three groups of anesthesia. PaO2 values on first postoperative day were significantly lower than those before operation, and PaO2 value in enflurane group (PaO2 = 67 +/- 1 mmHg) was significantly lower than that in halothane group (PaO2 = 72 +/- 2 mmHg, P less than 0.05).

L47 ANSWER 39 OF 55 MEDLINE
AN 89270963 MEDLINE
DN 89270963 PubMed ID: 2567127
TI [Postoperative, opiate-induced respiratory depression is not dependent on arousal].
Die postoperative, opiatbedingte Atemdepression ist nicht abhangig von der Vigilanz.
CM Comment in: Anasth Intensivther Notfallmed. 1990 Aug;25(4):297-300
AU Tolksdorf W; Bremer H; Tokic B
CS Klinik fur Anaesthesiologie, Med. Fak. der RWTH, Aachen.
SO ANASTHESIE, INTENSIVTHERAPIE, NOTFALLMEDIZIN, (1989 Apr) 24 (2) 94-9. Journal code: 8005775. ISSN: 0174-1837.
CY GERMANY, WEST: Germany, Federal Republic of
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA German
FS Priority Journals
EM 198906
ED Entered STN: 19900309
Last Updated on STN: 20020522
Entered Medline: 19890623

AB INTRODUCTION: Respiratory depression after intravenous anesthesia is supposed to be related to vigilance. This hypothesis could not yet be tested because of the lack of methods measuring continuously important parameters of respiration without altering the patient's vigilance. Pulse Oximetry offers this possibility. The following study was performed to investigate the effects of the Benzodiazepine Antagonist Flumazenil (Anexate) on parameters of vigilance and respiration after Midazolam/Fentanyl anesthesia. METHODS: 40 healthy patients aged 18-65 years who were to undergo arthroscopy were randomly assigned to Flumazenil (Group F) or no Flumazenil = Control (Group C). All patients received 7.5-10 mg Midazolam, 0.3-0.6 Fentanyl, 4-6 mg Vecuronium, were intubated and ventilated with 8 ml/kg BW VT x 12/min - **N2O**/O2 = 2:1. At the end of operation Group F received 0.3-0.5 mg Flumazenil. When adequate spontaneous ventilation was restored the patients were extubated and brought to a single bed room where they were monitored and observed without being disturbed, except at the arrival time (T1), 15 min (T2) and 30 min (T3) when blood pressure was measured and the pain score was asked. The following parameters were registered: Transcutaneous Oxygen Saturation (SAT) and Heart Rate (HR) continuously, Sedation (every minute) and Reactions to acoustic or verbal stimuli in the case of **hypoxemia** . The two groups were compared with respect to the number and severity of hypoxic events/15 min, the mean degree of sedation (6 point scale) and the number of adequate reactions to the acoustic alarm resp. instruction: "Take a deep breath!" STATISTICS: Wilcoxon Test, Chi-Square test (p less than or equal to 0.05) is significant). RESULTS: The groups were comparable with respect to their anthropometric data, dosages of Midazolam and Fentanyl, and perioperative blood pressures. Parameters of vigilance: Group F was less sedated than Group C (p = 0.052) and reacted better to the verbal instruction to take a deep breath in the case of **hypoxia** (p less than or equal to 0.05). Parameters of respiration: Hypoxic states occurred more frequently in group F (p = 0.098) and lasted longer. The severeness was significantly more pronounced in group F (p less than or equal to 0.05). There were no complications and the patients acceptance of the anesthetic procedure was high. CONCLUSIONS: The hypothesis that postoperative respiratory depression is related to the degree of vigilance cannot be accepted. In contrast there is a strong evidence that under special conditions patients can be in a relatively high degree of vigilance and do not breathe with subsequent severe **hypoxemia**. The possible u

L47 ANSWER 40 OF 55 MEDLINE
AN 83104130 MEDLINE
DN 83104130 PubMed ID: 6822071
TI Postoperative nitrous oxide analgesia and the functional residual capacity.
AU Kripke B J; Justice R E; Hechtman H B
NC 5 PO1-GM-17366-05 (NIGMS)
SO CRITICAL CARE MEDICINE, (1983 Feb) 11 (2) 105-9.
Journal code: 0355501. ISSN: 0090-3493.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198303
ED Entered STN: 19900318
Last Updated on STN: 19970203
Entered Medline: 19830324
AB Surgery of the upper abdomen is associated with the greatest demand for postoperative analgesia and also is marked by depressed pulmonary

function, arterial **hypoxemia**, and pulmonary complications. Nitrous oxide (**N2O**) in concentrations of 15-25% is a potent analgesic and is relatively free of untoward side effects if administered for a maximum of 48 h. In the present study, the effect of **N2O** analgesia on postoperative lung function, in particular, the functional residual capacity (FRC), is examined. Eighteen cholecystectomy patients received either a narcotic (N = 11) or **N2O** (N = 7) for postoperative analgesia. **N2O**-treated patients had satisfactory analgesia and maintained FRC at normal levels. Narcotic treated patients had a fall of 22% in FRC. **N2O** had no effect on the formed elements in peripheral blood.

L47 ANSWER 41 OF 55 MEDLINE
AN 77101742 MEDLINE
DN 77101742 PubMed ID: 834512
TI Sudden death in an infant from methemoglobinemia after administration of "sweet spirits of nitre".
AU Chilcote R R; Williams B; Wolff L J; Baehner R L
SO PEDIATRICS, (1977 Feb) 59 (2) 280-2.
Journal code: 0376422. ISSN: 0031-4005.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197703
ED Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770321
AB The administration of "sweet spirits of nitre" (4% **ethyl nitrite** CH3CH2ONO in 70% ethyl alcohol) was followed by acute methemoglobinemia and severe anoxic metabolic acidosis in infant twins, Methylene blue administration reversed methemoglobinemia in both, but one twin died from the consequences of **hypoxemia**. Hemoglobin electrophoresis and methemoglobin reductase determinations were normal for age. This medicine is available without prescription and contains the potent oxidant **ethyl nitrite**. In infants with sudded death or onset of cyanosis, appropriate toxicological and historical information should be obtained.

L47 ANSWER 42 OF 55 MEDLINE
AN 75089095 MEDLINE
DN 75089095 PubMed ID: 1111385
TI Circulatory effects of halothane and halothane-nitrous oxide anesthesia in the dog: spontaneous ventilation.
AU Steffey E P; Gillespie J R; Berry J D; Eger E I; Rhode E A
SO AMERICAN JOURNAL OF VETERINARY RESEARCH, (1975 Feb) 36 (2) 197-200.
Journal code: 0375011. ISSN: 0002-9645.
Report No.: NASA-75089095.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 197504
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19750423
AB The cardiovascular effects of equipotent (minimum alveolar concentration; MAC) doses of halothane versus halothane plus 25% **N2O** (H25N2O) in spontaneously breathing dogs do not differe except that nitrous oxide

increased mean arterial pressure (AP) and decreased arterial oxygen partial pressure (PAO2). When 75% nitrous oxide was added to halothane anesthesia, AP, mean pulmonary artery pressure (PAP), heart rate (HR), cardiac output (CO), stroke volume (SV), total peripheral resistance (TPR), and left ventricular work (LVW) increased and PAO2 and hemoglobin saturation decreased. Arterial oxygen tensions below 80 torr were common at moderate and deep anesthetic levels of halothane plus 75% **N2O** (H75N2O). The specific contribution of **N2O, hypoxemia,** hypercapnia, or temporal recovery (or a combination of these) in producing cardiovascular stimulation were not determined.

L47 ANSWER 43 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999417643 EMBASE
TI Volatile induction and maintenance (VIMA) versus total intravenous anaesthesia (TIVA) for minor gynaecological procedures.
AU Ong E.L.; Chiu J.W.; Chong J.L.; Kwan K.M.
CS E.L. Ong, Department of Anaesthesia, Surgical Intensive Care, Changi General Hospital, 2 Simei Street 3, 529889 Singapore, Singapore
SO Ambulatory Surgery, (2000) 8/1 (37-40).
Refs: 19
ISSN: 0966-6532 CODEN: AMSUF3
PUI S 0966-6532(99)00030-X
CY United Kingdom
DT Journal; Article
FS 010 Obstetrics and Gynecology
024 Anesthesiology
030 Pharmacology
037 Drug Literature Index
009 Surgery
LA English
SL English
AB We compared the techniques of volatile induction and maintenance (VIMA) and total intravenous anaesthesia (TIVA) in various aspects. Patients undergoing spontaneous respiration-general anaesthesia were randomised into two groups; Group P received iv fentanyl 1 .mu.g/kg and propofol 2 mg/kg for induction followed by propofol 10 mg/min as required. Group S received vital capacity induction with sevoflurane and were maintained on 66% **N2O** in O2 with sevoflurane 2%. Induction times, complications and recovery times were recorded. Visual analogue scores for pain and satisfaction were assessed. The two groups did not differ significantly in emergence times or VAS scores for pain and satisfaction but more complications like apnoea and injection pain were encountered during TIVA compared to VIMA. Our results suggest that both techniques are comparable in efficacy for providing anaesthesia in minor gynaecological surgery with swift induction, good recovery and minimal postoperative complications. Copyright (C) 2000 Elsevier Science B.V.
CT Medical Descriptors:
*anesthesia
*gynecologic surgery
apnea: EP, epidemiology
apnea: CO, complication
postoperative pain: EP, epidemiology
postoperative pain: CO, complication
hypoxemia: EP, epidemiology
hypoxemia: CO, complication
injection pain: EP, epidemiology
injection pain: CO, complication
recall
awareness

nausea: EP, epidemiology
nausea: CO, complication
patient satisfaction
human
female
major clinical study
intravenous drug administration
inhalational drug administration
clinical trial
randomized controlled trial
article
Drug Descriptors:
fentanyl: CM, drug comparison
fentanyl: CB, drug combination
propofol: CM, drug comparison
propofol: CB, drug combination
sevoflurane: CM, drug comparison
sevoflurane: CB, drug combination
nitrous oxide plus oxygen: CM, drug comparison
nitrous oxide plus oxygen: CB, drug combination

L47 ANSWER 44 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1998275315 EMBASE
TI [Recommendations of the neuroanesthesia working group of the German Society of Anaesthesiology and Intensive Care Medicine for the acute management of patients with severe head trauma].
INNERKLINISCHE AKUTVERSORGUNG DES PATIENTEN MIT SCHADEL-HIRN-TRAUMA: EMPFEHLUNGEN DES WISSENSCHAFTLICHEN ARBEITSHREISES NEUROANASTHESIE DER DGAI.
AU Dinkel M.; Hennes H.-J.
CS Dr. H.-J. Hennes, Klinik fur Anasthesiologie, Johannes Gutenberg-Universitat Mainz, Langenbeckstrasse 1, D-55131 Mainz, Germany
SO Anasthesiologie und Intensivmedizin, (1998) 39/7-8 (399-412).
Refs: 122
ISSN: 0170-5334 CODEN: ANIMD2
CY Germany
DT Journal; General Review
FS 009 Surgery
024 Anesthesiology
LA German
SL English; German
AB In the acute management of patients suffering from severe head trauma, it is the predominant task of the anesthesiologist to avoid secondary cerebral injury. Such secondary brain damage after trauma is caused by different intra- as well as extracranial influences and limits the prognosis. Main causes of secondary brain damage are arterial hypotension and **hypoxemia** which are inherent in 10 to 20% of the patients at the time of admission to the emergency room, despite preclinical measures. For the management of the patient with head trauma it is essential to keep up a sufficiently high cerebral perfusion pressure (CPP .ltoreq. 70 mmHg) in order to prevent ischemia during the early stage after the head trauma, during which cerebral blood flow is frequently impaired. As arterial hypotension is an independent determinant of an unfavourable neurologic result, even short-term hypotensive periods cannot be tolerated. In order to stabilise the circulatory system and increase CPP, isotonic electrolyte solutions and/or colloidal solutions are administered; eventually catecholamines may be indicated. Extracranial hemorrhage must be carefully looked for and its management is of great significance. To prevent secondary cerebral injury it is important to stabilize the circulatory

system on the one hand, and on the other to prevent **hypoxemia**, hypercapnia and extreme hypocapnia. Up to 70% of the patients suffering from severe head trauma initially show - apart from the **hypoxemia** - hypercapnia, which increases intracranial pressure (ICP) by cerebral vasodilatation. Conversely, forced hyperventilation (paCO2 < 30 mmHg) also adversely affects the neurologic result. Therapy is therefore aimed at normoxygenation (saO2 > 95%, paO2 > 100 mmHg) as well as normocapnia in the lower normal range (paCO2 .simeq. 35 mmHg, petCO2 .simeq. 30-32 mmHg). In case these criteria are not complied with on admission of the patient to the emergency room, the trachea must be intubated and the lungs be ventilated; monitoring shall include pulse oximetry, capnometry and repeated blood gas analyses. The same applies to all patients with eight or less points on the Glasgow Coma Scale (GCS) and those with an impaired oxygen transport capacity. Any increase of the intracranial pressure (ICP) caused by stress or pain must be prevented by analgosedation or anesthesia (titrated dosage, intravenous anesthetic agents, no **N2O**); stress protection is required even in comatose patients. Cerebral diagnostics is aimed at an early identification of the type, localisation and prognostic significance of intracranial lesions as well as the assessment of the efficacy of therapeutic measures. Apart from the repeated clinical examination, both cranial computed tomography (CCT) and ICP measurement are very helpful to accomplish these aims. Epidural and acute subdural hematomas must be evacuated without delay. The introduction of measures to lower ICP is indicated with an ICP of more than 20 mmHg. Therapy is aimed at a reduction of ICP and an increase of CPP. Basic measures to reduce ICP include hemodynamic stabilisation, oxygen, analgosedation and head elevation (.ltoreq. 30.degree.). Further supportive measures are drainage of CSF, administration of mannitol as a bolus, hyperventilation (paCO2 30-35 mmHg), barbiturate therapy, and mild hypothermia. Specific drugs for brain protection cannot be recommended at this time because there is no proof for their clinical efficacy (e.g. glucocorticoids) or the expected benefit is limited to special indications (such as calcium antagonists in traumatic subarachnoidal hemorrhage).

CT Medical Descriptors:
*intensive care
*head injury: SU, surgery
*brain injury: SU, surgery
*unconsciousness: CO, complication
*neuroprotection
*subarachnoid hemorrhage: CO, complication
*subarachnoid hemorrhage: SU, surgery
postoperative care
anesthesia induction
disease severity
patient care
disease control
hypoxemia: CO, complication
hypoxemia: PC, prevention
hypercapnia: CO, complication
hypercapnia: PC, prevention
hypocapnia: CO, complication
hypocapnia: PC, prevention
oxygen transport
brain perfusion
hyperventilation
stress
injury scale
prognosis
electrolyte blood level

human
review
Drug Descriptors:
mannitol

L47 ANSWER 45 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 97176696 EMBASE
DN 1997176696
TI Postoperative analgesia with parenteral opioids: Does continuous delivery utilizing a transdermal opioid preparation affect analgesic efficacy or patient safety?.
AU Sevarino F.B.; Paige D.; Sinatra R.S.; Silverman D.G.
CS Dr. F.B. Sevarino, Department of Anesthesiology, Yale University School of Medicine, 333 Cedar Street, New Haven, CT 06520-8051, United States
SO Journal of Clinical Anesthesia, (1997) 9/3 (173-178).
Refs: 20
ISSN: 0952-8180 CODEN: JCLBE7
PUI S 0952-8180(97)00043-3
CY United States
DT Journal; Article
FS 024 Anesthesiology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Study Objectives: To compare, in patients who underwent major orthopedic surgical procedures, the efficacy of intravenous (IV) patient-controlled analgesia (PCA) with morphine combined with continuous administration of two doses of fentanyl or placebo via transdermal therapeutic system with fentanyl (TTSF) patches. Design: Randomized, double-blind, placebo-controlled study. Setting: University teaching hospital. Patients: 62 patients aged 18 to 65 years, presenting for elective orthopedic surgery and general anesthesia. Interventions: Patients were randomized to one of three groups: group 1 received two placebo patches: group 2 received a 20 cm2 active patch delivering 50 .mu./hr of fentanyl and a 30 cm2 placebo patch; group 3 received a 30 cm2 active patch delivering 75 .mu./hr of fentanyl and a 20 cm2 placebo patch. All patches were placed approximately two hours prior to induction of general anesthesia. General anesthesia was induced with thiopenial, intubation facilitated by the use of vecuronium or pancuronium, and anesthesia was maintained with isoflurane in an oxygen/nitrous oxide mixture (O2/**N2O**).
Following surgery, IV morphine was provided using IV PCA with 1.5 mg of morphine with a 6-minute lockout and a 4-hour maximum dosage of 30 mg. Measurements and Main Results: The time and dosage of morphine administered was recorded. Vital signs, pain intensity at rest, level of sedation, and arterial oxygen saturation (SpO2) were measured at intervals throughout the 72-hour study period and at 6 and 12 hours following patch removal. The presence of side effects was noted. Visual analog pain scores throughout the 72 hours of the study were not significantly different among groups. Patients receiving active TTSF required less IV PCA morphine at all time intervals. However, total opioid consumption was comparable among groups. The incidence of side effects was similar in all groups. Conclusions: There is no significant advantage to the routine use of continuous transdermal opioid delivery in patients receiving IV PCA after major orthopedic surgery.
CT Medical Descriptors:
*postoperative pain: DT, drug therapy
*postoperative pain: CO, complication
adult

article
bradypnea: SI, side effect
clinical trial
controlled study
double blind procedure
drug efficacy
drug safety
female
gastrointestinal symptom: SI, side effect
general anesthesia
human
hypoxemia: SI, side effect
inhalational drug administration
intravenous drug administration
major clinical study
male
neurotoxicity: SI, side effect
orthopedic surgery
pain assessment: DI, diagnosis
patient controlled analgesia
postoperative analgesia
priority journal
pruritus: SI, side effect
randomized controlled trial
transdermal drug administration
urine retention: SI, side effect
Drug Descriptors:
*fentanyl: AE, adverse drug reaction
*fentanyl: PD, pharmacology
*fentanyl: DT, drug therapy
*fentanyl: CM, drug comparison
*fentanyl: CB, drug combination
*fentanyl: CT, clinical trial
*fentanyl: AD, drug administration
*morphine: DO, drug dose
*morphine: PD, pharmacology
*morphine: AE, adverse drug reaction
*morphine: CM, drug comparison
*morphine: CB, drug combination
*morphine: AD, drug administration
*morphine: CT, clinical trial
*morphine: DT, drug therapy
*opiate agonist: AE, adverse drug reaction
*opiate agonist: PD, pharmacology
*opiate agonist: DT, drug therapy
*opiate agonist: CM, drug comparison
*opiate agonist: CB, drug combination
*opiate agonist: AD, drug administration
*opiate agonist: CT, clinical trial
isoflurane
nitrous oxide
pancuronium
thiopental
vecuronium

L47 ANSWER 46 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 96071826 EMBASE
DN 1996071826
TI Intraoperative ventilation with air and oxygen during laparoscopic

cholecystectomy decreases the degree of postoperative hypoxaemia.
AU Fujii Y.; Tanaka H.; Toyooka H.
CS Department of Anaesthesiology, Toride Kyodo General Hospital, 2-1-1 Hongo,Toride City, Ibaraki 302, Japan
SO Anaesthesia and Intensive Care, (1996) 24/1 (42-44).
ISSN: 0310-057X CODEN: AINCBS
CY Australia
DT Journal; Article
FS 009 Surgery
024 Anesthesiology
048 Gastroenterology
LA English
SL English
AB We studied the effects of intraoperative use of air in oxygen (O2) (FiO2 = 0.33) versus nitrous oxide (**N2O**) in O2 (FiO2 = 0.33) on the degree of postoperative hypoxaemia in 30 patients undergoing laparoscopic cholecystectomy. Patients were randomly allocated to receive either general anaesthesia with air (Group A, n = 15) or with **N2O** (Group N, n = 15). Arterial gas tensions were measured before, 24 h and 48 h after surgery while breathing room air. The mean P(a)O2 24 h and 48 h postoperatively decreased significantly in both groups compared with the preoperative values. The mean P(a)O2 24 h postoperatively in Group N (74.6 .+-. 6.4 mmHg) fended to be lower than that in Group A (78.1 .+-. 8.3 mmHg). The mean P(a)O2 48 h postoperatively in Group N (75.0 .+-. 7.8 mmHg) was significantly lower than that in Group A (83.5 .+-. 7.9 mmHg) (P < 0.05). On the contrary, the mean P(a)CO2 did not show any significant change during 48 h postoperatively in either group. Our results suggest that ventilation with **N2O** and O2 during laparoscopic cholecystectomy is associated with a lower degree of postoperative hypoxaemia.
CT Medical Descriptors:
*assisted ventilation
*cholecystectomy
***hypoxemia: PC, prevention**
***hypoxemia: CO, complication**
adult
arterial carbon dioxide tension
arterial oxygen tension
article
clinical article
clinical trial
controlled study
female
general anesthesia
human
male
postoperative complication
randomized controlled trial
Drug Descriptors:
*oxygen
nitrous oxide

L47 ANSWER 47 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 94192642 EMBASE
DN 1994192642
TI Influence of **N2O** on pulmonary shunt during one-lung-anaesthesia in pigs.
AU Hartung H.-J.; Strauss K.M.; Kamner L.; Funk A.
CS Anaesthesio./Inten. Care Med. Inst., Krankenhaus Am Urban, Dieffenbachstr.

1,W-1000 Berlin, Germany
SO Journal of Cardiothoracic and Vascular Anesthesia, (1994) 8/3 SUPPL. 2 (178).
ISSN: 1053-0770 CODEN: JCVAEK
CY United States
DT Journal; Conference Article
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
024 Anesthesiology
037 Drug Literature Index
LA English
TI Influence of **N2O** on pulmonary shunt during one-lung-anaesthesia in pigs.
CT Medical Descriptors:
*hypoxic lung vasoconstriction
*inhalation anesthesia
*lung arteriovenous shunt
animal experiment
artificial ventilation
conference paper
hypoxemia: CO, complication
inhalational drug administration
lung vascular resistance
lung ventilation
nonhuman
priority journal
pulmonary hypertension: CO, complication
swine
Drug Descriptors:
*nitrous oxide

L47 ANSWER 48 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 90185472 EMBASE
DN 1990185472
TI Hyperbaric nitrous oxide as a sole anesthetic agent in humans.
AU Russell G.B.; Snider M.T.; Richard R.B.; Loomis J.L.
CS Department of Anesthesia, Milton S. Hershey Med. Cent., Pennsylvania State University, P.O. Box 850,Hershey, PA 17033, United States
SO Anesthesia and Analgesia, (1990) 70/3 (289-295).
ISSN: 0003-2999 CODEN: AACRAT
CY United States
DT Journal; Article
FS 024 Anesthesiology
037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
AB Nitrous oxide (**N2O**) has been used to produce analgesia and anesthesia for more than 100 yr. However, because of its high MAC value (1.04), general anesthesia with **N2O** can usually be attained only in a hyperbaric environment. Because of the sparsity of documentation for human physiologic responses to hyperbaric **N2O**, we studied eight male volunteers at 2 ATA (1520 mm Hg) anesthetized with **N2O** only for periods of 2-4 h. **N2O** partial pressures ranged from 836 to 1368 mm Hg. The anesthetic state was associated with tachypnea, tachycardia, increases in systemic blood pressure, mydriasis, diaphoresis, and at times, clonus and opisthotonus. A stable level of physiologic activity was difficult to maintain.
CT Medical Descriptors:
*general anesthesia

*hyperbaric chamber
*hypertension: SI, side effect
***hypoxemia**
*mydriasis: SI, side effect
*tachypnea: SI, side effect
adult
clinical article
human
male
inhalational drug administration
article
priority journal
Drug Descriptors:
*nitrous oxide: AE, adverse drug reaction
*nitrous oxide: PD, pharmacology
*nitrous oxide: AD, drug administration

L47 ANSWER 49 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 88163409 EMBASE
DN 1988163409
TI Should oxygen be administered after laporoscopy in healthy patients?.
AU Vegfors M.; Cederholm I.; Lennmarken C.; Lofstrom J.B.
CS Department of Anaesthesiology, University Hospital, S-581 85 Linkoping, Sweden
SO Acta Anaesthesiologica Scandinavica, (1988) 32/4 (350-352).
ISSN: 0001-5172 CODEN: AANEAB
CY Denmark
DT Journal
FS 024 Anesthesiology
LA English
SL English
AB This study aimed to assess the oxygen flow necessary to maintain satisfactory oxygen saturation when administered via a nasopharyngeal catheter. Oxygen saturation was displayed by a pulse oximeter and/or measured in arterial blood samples. Thirty-six healthy women scheduled for elective diagnostic laparoscopy were anaesthetized using thiopentone, fentanyl and O2/**N2O**. Atracurium was used as relaxant which was reversed with atropine and neostigmine. Arterial samples were obtained prior to anaesthesia, on arrival in the postoperative ward and 1 h postoperatively. Oxygen saturation was monitored postoperative using a pulse oximeter. The patients were randomly divided into three groups which received either no oxygen, 2 l O2/min or 4 l O2/min. On arrival in the postoperative ward 15% of the patients were below the normal limit of O2 saturation (94%). In patients receiving 2 l or 4 l O2, oxygen saturation was well above normal values. In patients receiving no oxygen, two had low oxygen saturation (92% and 93%). Comparing saturation values obtained in arterial samples with values measured with pulse oximetry gave r = 0.79. It is concluded that all patients should be given oxygen in the immediate postoperative period. Increasing oxygen flow from 2 to 4 l/min had no major effect on oxygen saturation. These results were obtained in healthy patients following minor abdominal surgery.
CT Medical Descriptors:
*anesthesia
*blood gas
***hypoxemia: PC, prevention**
*laparoscopy
*postoperative period
human experiment
human

normal human
inhalational drug administration
therapy
Drug Descriptors:
*oxygen

L47 ANSWER 50 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 88124104 EMBASE
DN 1988124104
TI Hypoxaemia following sustained low-volume venous air embolism in sheep.
AU Pfitzner J.; Petito S.P.; McLean A.G.
CS The Queen Elizabeth Hospital, Woodville, SA 5011, Australia
SO Anaesthesia and Intensive Care, (1988) 16/2 (164-170).
ISSN: 0310-057X CODEN: AINCBS
CY Australia
DT Journal
FS 002 Physiology
024 Anesthesiology
LA English
SL English
AB In six upright (head above thorax) anaesthetized sheep, serial blood gas measurements were made over a 100-minute period during which repeated small-volume air emboli were injected intravenously to lower and maintain the end-tidal CO2 concentration approximately 0.5% below its initial baseline level. With constant volume ventilation and an inspired **N2O**:O2 ratio of 2:1, the arterial PCO2 progressively increased and the arterial PO2 progressively decreased with significant arterial hypoxaemia ensuing in three out of the six animals. It is suggested that during neurosurgery performed in the sitting position and with an inspired oxygen concentration of 33%, the degree of cardio-respiratory disturbance caused by venous air embolism should be assessed by continuous monitoring not only of end-tidal CO2 concentration but also of arterial oxygen saturation using pulse oximetry.
CT Medical Descriptors:
*blood gas analysis
*cardiopulmonary disease
*embolism
***hypoxemia**
hemodynamics
sheep
methodology
animal experiment
nonhuman

L47 ANSWER 51 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 86083264 EMBASE
DN 1986083264
TI Anesthetic management for carinal resection.
AU Kobayashi S.; Kasama A.; Kaya K.
CS Department of Anesthesiology, Tokyo Metropolitan Komagome Hospital, Tokyo 113, Japan
SO Japanese Journal of Anesthesiology, (1986) 35/2 (312-317).
CODEN: MASUAC
CY Japan
DT Journal
FS 037 Drug Literature Index
015 Chest Diseases, Thoracic Surgery and Tuberculosis
024 Anesthesiology
030 Pharmacology

016 Cancer
020 Gerontology and Geriatrics
011 Otorhinolaryngology

LA Japanese

SL English

AB A 69 year old man with mucoepidermoid cancer at the carinal region, underwent resection of the carina, esophagus, upper lobe and S 6 region of the rt-lung and reconstruction of this area. An epidural catheter was inserted into the Th6-7 interspace and 6 mg morphine chloride in 15 ml of saline injected epidurally. Anesthesia was induced with thiopental 250 mg and diazepam 5 mg i.v., followed by pancuronium 4 mg i.v. and maintained with **N2O** (60%) in O2. During reconstruction of the carinal region, 3 types of ventilation were attempted. Under dependent lung ventilation with oxygen (FIO2 0.3) only, PaO2 decreased greatly and P-A-P- increased, but PaCO2 was maintained in normal ranges. Under dependent lung ventilation with oxygen (FIO2 0.3) and continuous positive pressure (10 cm H2O) with oxygen (FIO2 1.0) to the non-dependent lung, PaO2 recovered to the level with bilateral lung ventilation, PaCO2 and PAP did not increase. With only HFJV to the dependent lung with oxygen (FIO2 0.5), PaO2 decreased and PaCO2 increased. From these results, it was concluded that **hypoxemia** and hypercapnia were induced readily under one lung ventilation, but in addition to it, continuous positive pressure with oxygen to the non-dependent lung terminated these untoward reactions.

CT Medical Descriptors:
*anesthesia
*cancer
*endobronchial anesthesia
***hypoxia**
*drug therapy
*trachea ridge
*trachea surgery
high frequency ventilation
positive end expiratory pressure
priority journal
therapy
inhalational drug administration
intramuscular drug administration
intravenous drug administration
case report
human
respiratory system
larynx
Drug Descriptors:
*diazepam
*morphine
*nitrous oxide
*oxygen
*pancuronium
*thiopental

L47 ANSWER 52 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

AN 79040452 EMBASE

DN 1979040452

TI Binary diffusion coefficient: Theory, experimental assessment and its implications as a limiting factor of pulmonary gas exchange at depths.

AU Ohta Y.; Kodaka Y.

CS Dept. Physiol., Sch. Med., Tokai Univ., Kanagawa, Japan

SO Tokai Journal of Experimental and Clinical Medicine, (1977) 2/4 (235-242). CODEN: TJEMDR

CY Japan
DT Journal
FS 002 Physiology
015 Chest Diseases, Thoracic Surgery and Tuberculosis
027 Biophysics, Bioengineering and Medical Instrumentation
LA English
AB Binary gas diffusion has attracted considerable attention in the field of respiratory physiology since it seems to play some important role in alveolar gas exchange. Many predictive, theoretical and/or empirical formulae have been reported for calculation of the coefficient. First, we review these formulae briefly. Second, results of experiments using Schwertz and Brow's method are presented. Binary diffusion coefficients for He-H2O, N2-H2O, air-H2O, **N2O**-H2O, CO2-H2O and SF6-H2O observed were 0.9400, 0.2737, 0.2986, 0.1917, 0.1877 and 0.1420, respectively, which showed good agreement with those predicted by the Slattery and Bird equation for water vapor, except for He-H2O. A comparison between the tentatively calculated time for diffusive mixing in the lungs and data reported by Chouteau et al. on the development of **hypoxemia** at depths suggested a possible role of diffusive mixing in Chouteau **hypoxia**.

L47 ANSWER 53 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 78226438 EMBASE
DN 1978226438
TI A study of pathophysiology of post-operative **hypoxemia**.
AU Okutsu Y.
CS Dept. Anesthesiol., Yokohama City Univ. Sch. Med., Yokohama, Japan
SO Japanese Journal of Anesthesiology, (1977) 26/8 (855-863).
CODEN: MASUAC
CY Japan
DT Journal
FS 024 Anesthesiology
037 Drug Literature Index
015 Chest Diseases, Thoracic Surgery and Tuberculosis
LA Japanese
SL English
TI A study of pathophysiology of post-operative **hypoxemia**.
AB Patients studied were divided into three groups, 1) patients who were anesthetized with GOF (O2-**N2O**-Halothane) and received upper abdominal surgery, 2) patients who were anesthetized with GOF and received minor surgery of lower abdomen or extremities, and 3) patients who received spinal anesthesia and surgery of extremities. Blood gas tensions and lung volume, including closing volume (CV) and FRC, were measured on the day before the operation, and on the 3rd and 7th post-operative days. The slope of phase III on a single breath N2 washout curve, which is considered to be an index of intrapulmonary gas distribution, was calculated from the trace of N2 washout curve. In the post-operative period, lung volumes decreased in patients with upper abdominal surgery, but not with GOF anesthesia. Upper abdominal surgery resulted in a decrease in CV and an increase in the slope of phase III of N2 washout curve. Pa(O2) showed a proportional change with these parameters. However, because of a decrease in FRC, CC/FRC did not show any significant change even after upper abdominal surgery. On the basis of these observations, it was concluded that the main causes of post-operative **hypoxemia** were both decrease of FRC and increase in uneven distribution of inspired gas, but the former had much greater effect on Pa(O2) than the latter.
CT Medical Descriptors:
*anesthesia complication
*functional residual capacity

***hypoxemia**
*lung function
*pathogenesis
*postoperative complication
etiology
major clinical study
Drug Descriptors:
*halothane
*nitrous oxide
*oxygen

L47 ANSWER 54 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 77104908 EMBASE
DN 1977104908
TI Air embolism as a main cause of cardiovascular changes in total hip arthroplasty (Japanese).
AU Sato I.
CS Dept. Anesthesiol., Saitama Med. Sch., Saitama ken, Japan
SO Japanese Journal of Anesthesiology, (1976) 25/7 (692-696).
CODEN: MASUAC
DT Journal
FS 024 Anesthesiology
033 Orthopedic Surgery
020 Gerontology and Geriatrics
018 Cardiovascular Diseases and Cardiovascular Surgery
LA Japanese
AB Hypotension and cardiac arrest have been reported to occur during total hip arthroplasty using methylmethacrylate cement to fix implants to the femur. Absorption of the monomer of methacrylate has been raised as a cause in many experiments. In the present study in cats, by raising the femoral intramedullary pressure from 100 mmHg to 300 mmHg with a blood pressure cuff, the author recognized air bubbling from veins of laminectomy wounds with marked hypotension, ECG abnormality and lowering of PaO2. Administration of 75% **N2O** after the restoration of blood pressure and ECG produced a gradual decline of blood pressure. Intravenous administration of 1 ml/kg of air in the other series of cats showed similar effects on blood pressure, ECG and PaO2. 75% **N2O** added in the inspired gas after five minutes of air injecion showed no remarkable chages in blood pressure or ECG. In autopsy after the experiment, air bubbles in popliteal veins and pulmonary arteries were observed.
CT Medical Descriptors:
*air embolism
*heart arrhythmia
*hypotension
***hypoxemia**
*total hip prosthesis
theoretical study
cat
Drug Descriptors:
*bone cement
*methacrylic acid methyl ester

L47 ANSWER 55 OF 55 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 77020837 EMBASE
DN 1977020837
TI [Experimental determination of blood alcohol metabolism with dogs in haemorrhagic shock].
TIEREXPERIMENTELLE UNTERSUCHUNGEN UBER DEN BLUTALKOHOLABBAU IM

STANDARDISIERTEN HAMORRHAGISCHEN SCHOCK.

AU Kaufmann H.; Tausch D.; Harbauer G.; Wagner H.J.
CS Inst. Rechtsmed., Univ. Saarland, Homburg, Germany

SO Zeitschrift fur Rechtsmedizin, (1976) 77/2 (79-89).
CODEN: ZRMDAN
DT Journal
FS 037 Drug Literature Index
049 Forensic Science Abstracts
005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry
030 Pharmacology
009 Surgery
LA German

AB Ethanol at a dosage of 3 g/kg reduced body weight was injected i.v. into mongrel dogs resulting in a blood alcohol concentration of approximately 2.9 mg/ml. One hour after injection the dogs were anesthetized with halothane **N2O**/O2 and blood was withdrawn until the blood pressure was reduced to 40 mm Hg. This usually required removal of about 30 - 40% of the total blood volume. The resulting hemorrhagic shock was ascertained by monitoring blood pH, pCO2, pO2, lactate, pyruvate and blood electrolytes. A blood specimen for enzymatic alcohol determination (ADH) was obtained every 30 min over a period of 3 hr. Compared with equally dosed controls the dogs in hemorrhagic shock showed a significant ($p = 0.005$) reduction of the blood alcohol decay rate which is explained by the diminished blood flow through the liver and the **hypoxemic** metabolic situation in shock.